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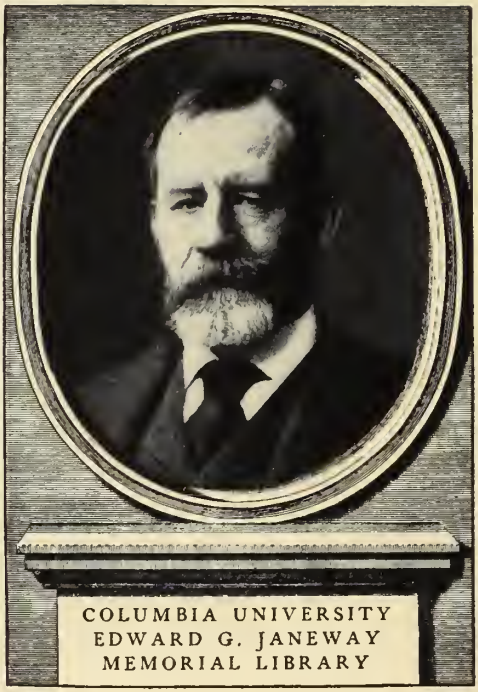

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TRANSACTIONS

OF THE

American Climatological and Clinical Association

FOR THE YEAR 1922

VOLUME XXXVIII

"The object of this Association shall be the study of Climatology and Hydrology and of Diseases of the Respiratory Organs."—*Constitution.*

PRINTED FOR THE ASSOCIATION
1922

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NOTICE.

The Association assumes no responsibility for the statements and opinions expressed in the papers read at its meetings.

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Judson Daland, M. D., Philadelphia.
J. H. Elliott, M.D., Toronto, Canada.
Guy Hinsdale, M.D., Hot Springs, Va.
Lawrason Brown, M.D., Saranac Lake, N. Y.
Carroll E. Edson, M.D., Denver, Colo.

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LIST OF OFFICERS.

Presidents.

Name	Year
A. L. Loomis	1884-5
William Pepper	1886
Frank Donaldson	1887
A. L. Loomis	1888
V. Y. Bowditch	1889
Charles Denison	1890
F. I. Knight	1891
W. E. Ford	1892
R. G. Curtin	1893
A. H. Smith	1894
S. E. Solly	1895
J. B. Walker	1896
E. Fletcher Ingals	1897
E. O. Otis	1898
Beverley Robinson	1899
Abraham Jacobi	1900
Robert H. Babcock	1901
Samuel A. Fisk	1902
Norman Bridge	1903
James C. Wilson	1904
W. F. R. Phillips	1905
E. L. Shurly	1906
Thomas Darlington	1907
Thomas D. Coleman	1908
Charles E. Quimby	1909
Edward R. Baldwin	1910
John Winters Brannan	1911
Alexander D. Blackader	1912

Name	Year
Charles L. Minor	1913
James M. Anders	1914
Henry Sewall	1915
James Alexander Miller	1916
Judson Daland	1917
Jabez H. Elliott	1918
Guy Hinsdale	1919
Lawrason Brown	1920
Carroll E. Edson	1921
William D. Robinson	1922

Vice-Presidents.

F. I. Knight, W. H. Geddings	1884-5
Frank Donaldson, Beverley Robinson	1886
V. Y. Bowditch, R. G. Curtin	1887
A. Y. P. Garnett, J. T. Whittaker	1888
J. R. Leaming, E. T. Bruen	1889
A. L. Gihon, H. B. Baker	1890
E. L. Trudeau, T. S. Hopkins	1891
E. Fletcher Ingals, Beverley Robinson	1892
A. H. Smith, E. O. Otis	1893
I. Hull Platt, E. L. Trudeau	1894
John H. Musser, G. R. Butler	1895
Charles E. Quimby, James A. Hart	1896
S. A. Fisk, John C. Munro	1897
Beverley Robinson, C. F. McGahan	1898
James A. Hart, R. C. Newton	1899
R. H. Babcock, J. W. Brannan	1900
Albert C. Peale, S. W. Langmaid	1901
Norman Bridge, W. F. R. Phillips	1902
James C. Wilson, H. S. Orme	1903
Thomas Darlington, Thomas D. Coleman	1904
S. G. Bonney, S. D. Risley	1905
A. D. Blackader, Henry Sewall	1906
Frank Fremont-Smith, C. L. Minor	1907
Judson Daland, Charles Fox Gardiner	1908
E. R. Baldwin, C. E. Edson	1909

Name	Year
James M. Anders, H. Longstreet Taylor	1910
Will Howard Swan, John H. Lowman	1911
Herbert Maxon King, Carroll E. Edson	1912
James M. Anders, C. D. Alton	1913
Lawrason Brown, Will Howard Swan	1914
Arthur K. Stone, James Alexander Miller	1915
Philip King Brown, Henry M. Bracken	1916
W. L. Dunn, Jabez H. Elliott	1917
W. G. Schauffler, H. M. Bracken	1918
Joseph H. Pratt, H. M. Kinghorn	1919
Charles W. Richardson, J. N. Hall	1920
Gordon Wilson, Estes Nicholson	1921
Col. George E. Bushnell, Charles D. Parfitt	1922

Secretaries and Treasurers.

James B. Walker	1884-95
Guy Hinsdale	1895-1918
Arthur K. Stone	1918-

LIST OF MEMBERS.

Honorary Members.

Elected

1888. Abbot, Griffith E., 9 Ocean Court, Manhattan Beach, Brooklyn, N. Y.
1914. Blue, Rupert, U. S. Public Health Service, Washington, D. C.
1914. Fox, R. Fortescue, 36 Devonshire Place, W., London.
1914. Gros, Edmund L., 23 Ave. du Bois de Boulogne, Paris, France.
1917. Marvin, Prof. Charles F., Chief, Weather Bureau, Washington, D. C.
1902. McBride, James H., Pasadena, Calif.
1914. Philip, Sir Robert W., Charlotte Sq., Edinburgh, Scotland.
O.M.* Robinson, Beverley, 129 East 35th St., N. Y.
1911. Stupart, Sir Robert F., K.B., F.R.C.S., Director, Dominion Meteorological Service, Toronto, Canada.
1898. Sunderland, Septimus, 11 Cavendish Pl., Cavendish Sq., W., London.
O.M.* Tyndale, J. Hilgard, 823 Terminal Bldg., Lincoln, Neb.
1917. Ward, Prof. R. De Courcy, Harvard Univ., Cambridge, Mass.
1907. Williams, Leonard L. B., 123 Harley St., W., London.
O.M.* Wilson, James C., 1509 Walnut St., Philadelphia.
1917. Wright, Sir Almroth, St. Mary's Hosp., London, England.

Corresponding Members.

1912. Carpenter, Ford A., Central Bldg., 6th and Main St., Los Angeles, Calif.
1898. Eyre, G. G., Claremont, Cape Town, South Africa.
1911. Grenfell, Wilfred, C.M.G., St. Anthony, Newfoundland.
1912. Henderson, Professor Yandell, 440 Prospect St., New Haven, Conn.
1908. Liston, Lieut.-Col. W. Glen, C.I.E., Indian Medical Service; Director, Bacteriological Lab., Bombay, India.

*O.M., Original Member.

Elected

1910. Rogers, Lt.-Col. Sir Leonard, C.I.E., F.R.S., F.R.C.S., Indian Medical Service, Prof. of Pathology, Medical College, Calcutta, India.
1902. Weber, F. P., 13 Harley St., W., London.
1914. Wood, Neville, 42 Elvaston Pl., Queen's Gate, London.

Life Members.

1897. Alton, Charles D., 1470 Asylum Ave., Hartford, Conn.
1915. Bailey, Walter C., Woodstock, Vt.
1897. Blackader, Alexander D., 236 Mountain St., Montreal, Can.
1895. Boardman, Wm. S., 63 Mt. Vernon St., Boston, Mass.
1894. Bridge, Norman, Los Angeles, Calif.
1897. Brown, Sanger, Kenilworth, Ill.
1896. Campbell, Wm. A., 424 N. Nevada Ave., Colorado Springs, Colo.
1889. Coolidge, A., 618 Beacon St., Boston, Mass.
1885. Ford, William E., Utica, New York.
1896. Gardiner, C. Fox, 1112 North Cascade Ave., Colorado Springs, Colo.
1892. Gibson, W. M., 260 Genesee St., New York .
1893. Hance, Irving C., Lakewood, N. J.
1891. Hart, James A., El Paso Club, Colorado Springs, Colo.
1896. Heffron, John L., 608 East Genesee St., Syracuse, N. Y.
1887. Platt, Walter B., 802 Cathedral St., Baltimore, Md.
1885. Rice, C. C., 125 E. 19th St., New York.
1890. Rogers, E. J. A., 222 Colfax Ave., Denver, Colo.
1887. †Smith, Fremont, Winter Park, Fla.
1898. Whitney, Herbert B., 320 Temple Court, Denver, Colo.
1898. Williams, Harold, 528 Beacon St., Boston, Mass.

Active Members.

1921. Adams, Roy D., Stoneleigh Ct., Washington.
1914. Alsever, William D., 727 East Willow St., Syracuse, N. Y.
1899. Anders, James M., 1605 Walnut St., Philadelphia.
1922. Amberson, James Burns, Loomis, N. Y.
1900. Arnold, Horace D., 520 Commonwealth Ave., Boston, Mass.
1893. Babcock, Robert H., 453 Deming Pl., Chicago.

†Died Sept. 1922.

Elected

1917. Baetjer, Walter A., 1400 Continental Bldg., Baltimore.
1898. Baldwin, Edward R., Saranac Lake, New York.
1901. Barlow, W. Jarvis, Security Bldg., Los Angeles, Calif.
1907. Barnes, Harry Lee, Wallum Lake, R. I.
1902. Bergtold, William H., 910 Metropolitan Bldg., Denver, Col.
1922. Blake, Gerald, 311 Beacon St., Boston, Mass.
1897. Bonney, S. G., Metropolitan Bldg., Denver, Col.
1885. Bowditch, V. Y., 506 Beacon St., Boston.
1891. Brannan, John W., 48 West 51st St., New York.
1919. Bray, Harry A., Ray Brook, New York.
1903. Brown, Lawrason, 104 Main St., Saranac Lake, New York.
1903. Brown, Philip King, Medical Bldg., San Francisco, Calif.
1907. Browning, Charles C., Merritt Bldg., 8th and Broadway, Los Angeles, Calif.
1919. Bruns, Earl A., Denver, Col.
1909. Bushnell, Col. George E., Bedford, Mass.
1886. Butler, G. R., 226 Gates Ave., Brooklyn.
1914. Byers, John Roddick, Ste. Agathe des Monts, P. Q., Canada.
1919. Chadwick, H. D., Westfield, Mass.
1918. Clark, Raymond, 310 Clinton Ave., Brooklyn, N. Y.
1906. Claytor, Thomas A., 1826 R St., N. W., Washington, D. C.
1901. Cobb, J. O., U. S. P. H., Chelsea, Mass.
1922. Cocke, Charles Hartwell, Asheville, N. C.
1894. Coleman, Thomas D., 986 Hickman Rd., Augusta, Ga.
1901. Collins, Charles Farnham, 830 Park Ave., New York City.
1917. Craig, Frank A., 2011 Pine Street, Philadelphia.
1910. Crane, Bayard T., Rutland, Mass.
1918. Crankshaw, Charles W., care of The Prudential Life Ins. Co., Newark, N. J.
1892. Daland, Judson, 317 South 18th St., Philadelphia.
1890. Darlington, Thomas, 27 Washington Square, N., New York City.
1907. Darnall, Wm. Edgar, 1704 Pacific Ave., Atlantic City.
1920. Dever, Francis J., 3rd and Cherokin Sts., Bethlehem, Pa.
1922. Kennon, Dunham, 1020 Union Central Bldg., Cincinnati, O.
1905. Dunn, William LeRoy, Asheville, N. C.
1897. Edson, Carroll E., 517 Majestic Bldg., Denver, Col.

Elected

1915. Elliott, John Barnwell, Jr., 1423 Louisiana Ave., New Orleans, La.
1903. Elliott, J. H., 11 Spadina Rd., Toronto, Canada.
1912. Farrand, Livingston, Ithaca, New York.
1910. Floyd, Cleaveland, 246 Marlborough St., Boston, Mass.
1917. Forster, Alexius M., Colorado Springs, Col.
1911. Fulton, Frank Taylor, 273 Bowen St., Providence, R. I.
1919. Garvin, Alfred H., 233 St. Antoine St., Detroit.
1915. Goodman, Edward H., 248 South 21st Street, Philadelphia.
1922. Gordon, Alvah H., 583 Dorchester St. W., Montreal, Can.
1921. Gorman, L. Whittington, Albany, N. Y.
1909. Griffin, Walter Alden, Sharon, Mass.
1907. Hall, J. N., 266 Metropolitan Bldg., Denver, Col.
1912. Hamman, Louis V., 1107 St. Paul St., Baltimore.
1922. Harlow, Wm. Page, Boulder, Colorado.
1922. Hart, William M., Daly Bldg., Ottawa, Can.
1912. Hatfield, Charles J., Montgomery Ave., Chestnut Hill, Philadelphia.
1914. Hawes, John B., 2d, 11 Marlboro St., Boston, Mass.
1918. Heise, Fred H., Trudeau, New York.
1893. Hinsdale, Guy, Hot Springs, Va.
1902. Hoagland, Henry W., Colorado Springs, Col.
1914. Holden, G. Walter, Agnes Memorial Sanatorium, Denver, Col.
1901. Jennings, Charles Godwin, 987 Jefferson Ave., Detroit, Mich.
1915. Johnston, Collins H., Metz Bldg., Grand Rapids, Mich.
1907. Kinghorn, Hugh M., 14 Church St., Saranac Lake, N. Y.
1899. Klebs, Arnold C., Nyon, Switzerland.
1922. Klein, Thomas, 1717 Pine St., Philadelphia, Pa.
1917. Klotz, Walter C., Johnson City, Tenn.
1921. Krause, Allen K., Johns Hopkins Hosp., Baltimore, Md.
1910. Landis, H. R. M., 2000 Pine St., Philadelphia.
1914. Lee, Roger I., Lowell St., Cambridge, Mass.
1914. Lichty, John A., 4634 Fifth Ave., Pittsburgh, Pa.
1918. Litchfield, Lawrence, 5111 Jenkens Arcade, Pittsburgh, Pa.
1917. Lloyd, John, 183 Vassar St., Rochester, N. Y.
1909. Locke, Edwin Allen, 311 Beacon St., Boston.

Elected

1921. Loomis, Philip A., Colorado Springs, Col.
1913. Lord, Frederick T., 305 Beacon St., Boston, Mass.
1907. Lyman, David Russell, Wallingford, Conn.
1918. McLean, John D., Asst. Health Commissioner, Harrisburg, Pa.
1921. McLester, James S., 930 So. 20th St., Birmingham, Ala.
1906. Manges, Morris, 72 East 79th St., New York.
1902. Marvel, Philip, 1616 Pacific Ave., Atlantic City, N. J.
1917. Matson, Ralph C., Corbett Bldg., Portland, Ore.
1917. Matson, Ray W., Corbett Bldg., Portland, Ore.
1917. Meader, Charles N., 518 Majestic Bldg., Denver, Col.
1915. Mercur, Wm. H., 5244 Fifth Ave., Pittsburgh, Pa.
1922. Metzger, Jeremiah, 1400 N. First Ave., Tucson, Arizona.
1919. Miller, A. F., Kentville, N. S.
1905. Miller, James Alexander, 379 Park Ave., New York.
1909. Miner, Charles H., 115 So. Franklin St., Wilkes-Barre, Pa.
1899. Minor, Charles L., Asheville, N. C.
1917. Montgomery, Charles M., U. S. P. H. Service, Oteen, N. C.
1919. Mount, W. B., Montclair, N. J.
1921. Musser, John H., Jr., 262 South 21st St., Philadelphia, Pa.
1907. Nichols, Estes, 1 Deering St., Portland, Me.
1917. Niles, Walter L., 893 Park Ave., New York.
1912. Norris, George W., 1820 Rittenhouse Sq., Philadelphia, Pa.
1888. Otis, E. O., 381 Beacon St., Boston, Mass.
1921. Packard, Edward N., Saranac Lake, New York .
1913. Parfitt, Charles D., Gravenhurst, Ontario, Canada.
1921. Paulin, James E., Docton Bldg., Atlanta, Ga.
1906. Perkins, Jay, 125 Waterman St., Providence, R. I.
1895. Phillips, W. F. R., School of Medicine, Charleston, S. C.
1917. Piersol, George Morris, 1913 Spruce St., Philadelphia.
1917. Porter, George D., 162 Crescent Rd., Toronto, Canada.
1922. Porter, William Branch, Lombardy and Franklin Sts., Richmond, Va.
1902. Pottenger, F. M., 1100 Title Insurance Bldg., Los Angeles, Calif.
1905. Pratt, Joseph H., 317 Marlborough St., Boston, Mass.

Elected

1917. Price, J. Woods, Saranac Lake, N. Y.
1922. Pritchard, J. S., Battle Creek, Mich.
1914. Probst, Charles O., 185 State St., Columbus, Ohio.
1905. Pryor, John H., 26 Linwood Ave., Buffalo, N. Y.
1919. Rathbun, Walter L., Lake Kushaqua, N. Y.
1901. Richardson, Charles W., 1317 Connecticut Ave., Washington, D. C.
1917. Ringer, Paul H., Asheville, N. C.
1890. Robinson, Wm. Duffield, 2012 Mt. Vernon St., Philadelphia.
1902. Rochester, Delancey, 469 Franklin St., Buffalo, N. Y.
1920. Ruffin, Sterling, The Connecticut, Cor. Connecticut Ave. and M St., Washington, D. C.
1917. Russell, Nelson G., 135 Linwood St., Buffalo, N. Y.
1918. Sawyer, John P., Rose Bldg., Cleveland, O.
1905. Schauffler, William Gray, Princeton, N. J.
1901. Sewall, Henry, 1360 Vine St., Denver, Col.
1915. Shattuck, George Cheever, 135 Marlborough St., Boston, Mass.
1910. Shurly, B. R., Adams Ave., W., Detroit, Mich.
1917. Smith, Wm. H., 8 Marlborough St., Boston, Mass.
1911. Steiner, Walter R., 646 Asylum Ave., Hartford, Conn.
1909. Stevens, Martin L., Asheville, N. C.
1919. Stewart, David A., Ninette, Manitoba.
1904. Stone, Arthur K., Auburn St., Framingham Center, Mass.
1915. Stone, Willard J., Pasadena, Calif.
1910. Swan, John M., 457 Park Ave., Rochester, N. Y.
1901. Swan, Will Howard, 20 E. San Rafael St., Colorado Springs, Col.
1892. Taylor, H. Longstreet, 814 Lowry Bldg., St. Paul, Minn.
1907. Taylor, J. Gurney, 330 Wells Bldg., Milwaukee, Wis.
1896. Taylor, J. Madison, 1504 Pine St., Philadelphia, Pa.
1910. Thomas, John D., The Farragut, N. W., Washington, D. C.
1920. Trudeau, Francis B., Saranac Lake, N. Y.
1919. Twitchell, David C., 25 Main St., Liberty, N. Y.
1917. Waters, Bertram H., Loomis, Sullivan Co., N. Y.
1917. Webb, Gerald B., Colorado Springs, Col.

Elected

1911. White, William Charles, Bedford Ave. and Wandless St., Pittsburgh, Pa.
1922. White, Paul D., Mass. General Hospital, Boston, Mass.
1885. Williams, H. F., 416 Grand Ave., Brooklyn, N. Y.
1911. Williams, Linsly R., 370 Seventh Ave., New York.
1910. Wilson, Gordon, 4 East Preston St., Baltimore, Md.
1917. Wilson, Robert, Jr., 165 Rutledge Ave., Charleston, S. C.
1913. Wood, Nathaniel K., 520 Beacon St., Boston, Mass.

MINUTES.

The 39th Annual Meeting of the American Climatological and Clinical Association was held in Washington, as a part of the Congress of American Physicians and Surgeons, on May 2nd, 3rd and 4th, 1922.

The meeting, under the leadership of Dr. William Duffield Robinson of Philadelphia, was probably one of the best attended meetings of the Association. Ninety-eight members were present at some part of the session.

The program was long and varied and was unusually interesting. Many of the guests expressed their pleasure at being present at the meetings of the Association, and said that they had elected to attend the meetings after a careful study of the whole program as presented by the Congress, and that they were more than glad they made the choice because of the character of the papers and of the discussion which had ensued.

At the annual dinner eighty-seven persons sat down at the tables in the Metropolitan Club and the speakers of the evening were Dr. Frank Billings, President of the Congress, who took the place of Dr. Vincent who was absent on account of sickness; Admiral Breasted; Dr. Darlington; and Charles W. Richardson the new President.

Under the able leadership of Drs. Hawes and Ringer an impromptu glee club sang most acceptably.

At the invitation of Capt. Myers of the Walter Reed Hospital there was a demonstration of the Sound Amplifier at the Signal Service Building, which was attended by many of the members.

Business.

The following officers were elected for the ensuing year:

President, Charles W. Richardson of Washington.

Vice-Presidents, Geo. W. Norris of Philadelphia, Will Howard Swan of Colorado.

Secretary and Treasurer, Arthur K. Stone of Framingham.

Recorder, Cleaveland Floyd of Boston.

Council, J. H. Elliott, Guy Hinsdale, Lawrason Brown, Carroll E. Edson, Wm. Duffield Robinson.

It was announced that the following members of the Society had died during the year: Charles E. Quimby, Robert C. Paterson, A. J. Richer, A. G. Shortle, Chas. E. Smith.

The Council recommended, and it was so voted by the Association, that the resignation of W. A. Jayne of Denver and the request of transference to life membership of Walter C. Bailey and Herbert B. Whitney be accepted. The following names of those who have been members of the society for 25 years or more were, at their request, transferred to life membership—Drs. Wm. S. Boardman, John L. Heffron, Jas. A. Hart, C. F. Gardiner, Irving H. Hance, Wm. A. Campbell, Sanger Brown, and Walter C. Platt.

All the names presented for membership by the Council were elected by the Association, namely:

1. H. Kennon Dunham, Cincinnati, Ohio.
2. Jeremiah Metzger, Tucson, Ariz.
3. William Page Harlow, Boulder, Colo.
4. Charles Hartwell Cocke, Asheville, N. C.
5. Thomas Klein, Philadelphia, Pa.
6. Gerald Blake, Boston, Mass.
7. Alvah H. Gordon, Montreal, Can.
8. Paul Dudley White, Boston, Mass.
9. William M. Hart, Ottawa, Can.
10. J. Stewart Pritchard, Battle, Creek, Mich.
11. James Burns Amberson, Loomis, N. Y.
12. William Branch Porter, Richmond, Va.

It was voted that a special assessment of $5.00 per member should be levied to enable the Association to print its Transactions up to date.

The following list of candidates for membership in the Association were presented and read at the meeting:

1. Horace John Howk, Mt. McGregor, N. Y.
2. L. J. Moorman, Oklahoma City, Okla.
3. John W. Flynn, Prescott, Ariz.
4. H. L. Goldstein, Philadelphia, Pa.

5. David Townsend, Johnson City, Tenn.
6. Edgar Mayer, Saranac Lake, N. Y.
7. Philip Norman, New York.
8. Charles G. Darlington, New York.
9. Thos. C. Kelley, Philadelphia, Pa.
10. W. B. Soper, Saranac Lake, N. Y.
11. H. M. Rich, Detroit, Mich.
12. H. C. Parsons, Toronto.

Nomination for honorary membership: Prof. Rollier of Switzerland.

The members are earnestly requested to look over this list carefully and to write to the Secretary *at once* their knowledge regarding any candidate that they may know in the above list. It is only by having the frank expression of opinion of the members that the Council can form an intelligent opinion as to the men which should be presented to the Association. Occasionally at the meetings of the Association the Council hears criticisms of its action regarding certain candidates whom it has recommended, when up to that time, everything had been favorable to the qualifications of the man in question. It is only by the active co-operation of all the members that the Council can do its best work for the Association.

Dr. Hinsdale presented an amendment to the constitution calling for an addition of two members to the Council. This amendment will be presented to the Council at its next meeting and reported on for action by the Association at its next annual meeting.

Telegrams were sent by the Association to the three original members now living, all of whom were unable to be present on account of illness.

The thanks of the Association were voted to the President for his energy and interest in the conduct of the meetings, and to Dr. Claytor, Chairman of the Committee on Arrangements.

Adjourned.

Arthur K. Stone,
Secretary.

MEMORIAL.

CHARLES E. QUIMBY, M.D.
1853-1921

Dr. Charles Elihu Quimby, a member of this Association and its president in 1909, died at his residence in New York City on November 6, 1921, from cerebral hemorrhage, in his 69th year.

Dr. Quimby was born in New Ipswich, New Hampshire in 1853 and was the son of Elihu Thayer Quimby and Mary Aldana Cutler. He was educated at the Appleton Academy of which his father was principal, entered Dartmouth College where his father had been appointed professor of mathematics and received his A.B. degree in 1874. Quimby then became principal of the high school in Gardner, Massachusetts, and the following year entered Dartmouth Medical College as the pupil of the brilliant teacher, Dr. A. Ben Crosby. He received from Dartmouth the degree of A.M. in 1877. He went to New York to complete his medical studies and received his M.D., with honor, in the University of the City of New York in February 1878.

At Bellevue Dr. Quimby was closely associated with Dr. Alfred L. Loomis to whom he was assistant for eleven years, in teaching physical diagnosis, and at the meeting in Hot Springs, Va., he read the beautiful memorial of Dr. Loomis which was published in our Transaction in 1895.

Besides his presidential address on The Elements of Nationality in Medical Science, Dr. Quimby's contributions to our transactions included papers on The Pneumatic Cabinet in the Treatment of Pulmonary Phthisis, The Mechanical Treatment of Heart Disease, The Definite Use of Physical Signs, The Treatment of Heart Disease and the discussion of various papers relating to tuberculosis and heart disease; the influence of the bicycle on the heart; and the effect of pneumatic differentiation on the heart.

Other papers included:

The Applied Physics of Physical Diagnosis. Medical Record, March 25, 1893.

A Factor in the Etiology of Distorted Nasal Septa. Medical News, March 12, 1904.

Pneumotherapy in Pulmonary Haemorrhage. N. Y. M. J., August 1, 1896.

The President's Address Before the Society of Alumni of Bellevue Hospital. N. Y. M. J., January 13, 1920.

The Relation of Intestinal Toxemia to Arteriorenal Disease. N. Y. State Jl. of Med., April 1902.

The Treatment of Bronchial Hemorrhage. Trans. Amer. Climat. Assoc., 1896.

On September 28th, 1881 Dr. Quimby was married to Miss Julia M. Cobb of Hanover, N. H. They had two children, Aldana Ripley, Born June 20th, 1888 and Dorothy Marian, born February 21st, 1892.

Dr. Quimby Was a member of the New York Academy of Medicine, the American Medical Association, president in 1899 of the Society of Alumni of Bellevue Hospital, and trustee of the Mary Hitchcock Hospital, Hanover, N. H. He was a member of the Alpha Delta Phi fraternity and club of New York and served as president from 1902 to 1904, having been a member of the Council for fifteen years. He served his fraternity with the utmost devotion.

Dr. Quimby inherited a mathematical mind and was consequently a logical thinker. His life was marked by a constant and untiring search for truth, in religion, medicine, and all sciences. He always believed that medicine was an exact science and that disease was not a fortuitous disturbance varying in the principles with the individual but that it was the result in all cases of a fixed process of scientific laws. Failure to diagnose was failure to reason not necessarily just lack of knowledge.

To all men he gave himself unsparingly, holding nothing back, never thinking of himself, seeking to serve wherever service might be given.

G. H.

ROBERT CHILDS PATERSON
1878-1921

The Christmas Season in the Village of Saranac Lake is an uncommonly joyous time. The very location of the place in the heart of the North Woods, with its pointed firs and deep snows makes it easy for reindeer to travel, and Christmas Eve of 1921 was no exception. There was a Community Tree in the Public Square —Santa Claus was surely coming—the streets were filled with children and their parents on their way to meet him, when suddenly the news that Doctor Paterson had been killed by the bullet of an insane man, was spread through the village,—leaving a wife and two little children in miserable sorrow, his medical colleagues bereft of one of their most beloved associates, and the whole community mystified.

Born in Montreal in 1878, he received his preliminary education in the public schools of that city. Was a graduate in Arts and Medicine of Magill University, being a gold medallist in both departments. After his graduation in Medicine in 1902, he served for two years as house physician and medical superintendent of the Montreal General Hospital, after which he had two years of post-graduate work in Europe, studying mainly in London, Greiburg, Berlin and Berne. In 1906 he returned to Montreal and was continuing the study of Pathology when he developed tuberculosis and was sent to Saranac Lake. There he regained his health and met Miss Winifred Marson, also of Montreal, whom he married in 1909, and moved to Ste. Agathe des Monts, Quebec, where he began the practice of medicine, specializing in tuberculosis. Feeling the lack of the stimulus of professional association in this isolated district, he returned to Saranac Lake in 1915, and there he worked until the day of his tragic death.

Paterson was well equipped for the work that he found there to do, and divided his time between research work in the Saranac Laboratory, and the practice of medicine in the Village, and was eminently successful in both fields. He was a frequent contributor to the medical journals, wrote the section on tuberculosis in Tice's "Practice of Medicine," and various other clinical and laboratory papers. His "The Pleural Reaction to Inoculation with Tubercle Bacilli in Vaccinated and Normal Guinea Pigs," read before this

Society and published in the American of Tuberculosis, was an especially valuable contribution to medical literature.

He was greatly interested in the surgical treatment of pulmonary and intestinal tuberculosis in cases that had failed to respond to routine treatment, and his frequent reports on an ever lengthening list of these cases did much to stimulate this work.

He was a lecturer in the Trudeau School of Tuberculosis, one of the visiting physicians to the Reception Hospital and the Saranac Lake General Hospital, and an active worker in various civic affairs of the Village.

His big frame, confident manner, hearty laughter and tender sympathy made him a welcome visitor to the sick porch. His untiring efforts to relieve their suffering without sacrifice of the utmost improvement that could be expected, and his close attention to the smallest detail of their illness, engendered a strong confidence and unusual devotion in his patients, who since his death have raised in his memory a trust fund, the income of which is to be applied to the help of needy cases.

He had an abiding faith in the higher and spiritual things, and was keenly interested in the church, being a vestryman and Superintendent of the Sunday School in the Church of St. Luke, The Beloved Physician.

He was a lover of outdoor sports, and took part in all that his physical handicap would allow. Golf and curling, ancient games of his Scotch ancestors, were his particular hobbies. Perhaps the greatest disappointment of Paterson's life was his repeated rejection on physical grounds by the medical examiners of the Canadian Army. Among the first to seek enlistment in 1914, he continued to apply at every lowering of the physical standard.

He was an interested member of the Climatological and Clinical Association from the time of his election in 1912, and helpful in his discussion of many medical problems that came before its sessions.

A devoted husband and father, a loyal friend, a good physician, Paterson's strong personality made a lasting impression an all with whom he came in contact.

J. W. P.

DR. A. J. RICHER.

Dr. Elliott. Mr. President and gentlemen, I only learned of his death three or four days ago in a medical journal.

In his younger days he was a very, very active in tuberculosis, but of late his health was not good, and he has had comparatively little practice. This is a note as published in the journal. His death occurred in his 54th year. On returning from Europe he devoted himself principally to tuberculosis. He was a pioneer in developing and establishing the first sanitorium in Quebec for the treatment of tuberculosis, and later for the treatment of patients, young women, recovering from acute respiratory diseases. He was actively connected with the Anti-Tuberculosis Association, and was a man who was particularly well liked by those of his patients to whom he devoted himself. In spite of his many peculiarities he was a man particularly fond of the woods, and often took his fishing rod and would disappear for days at a time, yet he had a very strong attraction for his patients, and many of them are sorry he is no longer with them.

DR. A. G. SHORTLE.

Dr. A. G. Shortle, a pioneer in the development of Albuquerque as a health resort and a national figure in tuberculosis work, died at 10.45 o'clock on May 26, 1922, at the Presbyterian sanatorium where he had been lying critically ill for several weeks. He had suffered from septicaemia since the middle of April, although he had not been in good health since January of this year.

The death of Dr. Shortle removes from Albuquerque a character who has had a big part in the development of the city. He was the founder of the local Rotary club. He was prominent in the fight which led to the adoption of the commission form of government in this city. He established the first sanatorium here for the treatment of tuberculosis. He was the prime mover in the reorganization in 1917 of the old commercial club into the present chamber of commerce.

In state affairs, he was nearly as well known, if the demands of his profession did not permit him to be as active. He was a strong advocate of the creation of a state department of health and was the

first physician to qualify as a member of that board. He retained his position when the board was merged into the department of public welfare and served as secretary of the enlarged department, declining the presidency when his vote would have made his election possible.

As a physician, particularly a tuberculosis specialist, his talents were recognized nationally. He was a member of the National Tuberculosis Association and served as a member of the special committee on migratory consumptives appointed by that body. He belonged to the American Climatological and Clinical Society and similar organizations.

Dr. Shortle was born in Rockville, Indiana, on February 14, 1871. He was graduated from the Northern Indiana Normal school in 1887, received an M.D. degree from the Baltimore Medical College in 1896, studied in the post-graduate medical school of the University of Chicago several summers and at the University of Heidelberg in Germany.

He was married to Alice Stanley Mitchell, of Chicago, on August 18, 1904. Four children were born to them—Margaret, Alice, Sarah and Samuel.

Dr. Shortle began the practice of medicine at Yorktown, Ill., in 1897 and practiced in Chicago from 1898 to 1905. He came to Albuquerque in 1908 and launched his sanatorium here, at that time encountering the opposition of the business men who thought it would injure the town. His personal efforts were largely responsible for starting the stream of healthseekers to Albuquerque, as he devoted much time to tours and lectures and wrote a number of articles.

Dr. Shortle's recent ill health began after a trip to Denver from which he returned on January 10. While he was up and around after the trip he lacked energy and appeared to have aged after his sickness. He made a trip to El Paso early in April and after his return he again went to bed, running a high fever and suffering from swollen glands. A week later he was removed to the Presbyterian sanatorium where he remained until his death.

DR. CHARLES EASTWICK SMITH, JR.

Dr. C. E. Smith, Jr. died on July 30, 1921. He was born in St. Paul in 1882, the son of Dr. Charles Eastwick Smith and grandson of Dr. Franklin Smith, who began his practice in St. Paul in 1855. Dr. Smith graduated at Yale in 1904. He then spent two years in the Medical School of the University of Minnesota, completing his work in the University of Pennsylvania, where he graduated in medicine in 1908.

Dr. Smith was always interested in public health work and from 1914 to the time of his death he was doing public health work either for the city or for the state. He eventually became the executive secretary of the Minnesota State Board of Health.

In 1909 he married Esther McDavitt, the daughter of Dr. Thomas McDavitt of St. Paul. She and three children survive him.

Never of robust build, an attack of pericarditis which he sustained in 1911 appears to have impaired his heart. A condition of bronchiectasis and emphysema gradually developed, and although sanitarium treatment for several months in 1917 appeared to be of benefit, his activities during the last few years were seriously interrupted. Several attacks of pneumonia increased the gravity of his condition, and in the hope of betterment of health, some time was spent in the southwest, but without avail. The common impression that the cause of death was pulmonary tuberculosis is entirely erroneous.

During these latter years of his life, he showed the stuff he was made of in the unfailing courage and energy with which he faced the world and went on with his work. During these years he was painfully short of breath, and although he would stop to rest a dozen times if he had to mount a flight of stairs, he bravely kept up the conversation and contrived to ignore the stops. Neved did he utter a complaint or rail at his hard lot although he accomplished more work than many men in good health would have done.

H. L. TAYLOR.

CONSTITUTION AND BY-LAWS.*

CONSTITUTION.

Article I.—Name.

This Society shall be known as the American Climatological and Clinical Association.

Article II.—Object.

The object of this Association shall be the Clinical Study of *Disease, especially of the Respiratory and Circulatory Organs, and of Climatology and Hydrology.*

Article III.—Membership.

Section 1.—This Association shall consist of *active, life, corresponding* and *honorary* members, the former not to exceed 150.

Section 2.—Names of candidates for active membership, whose applications shall have been endorsed by *three* (3) active members, shall be sent to the Secretary at or before the annual meeting at the second business session of which they shall be read and then lie over until the next annual meeting, when such as are approved by the Council shall be balloted on. Three (3) black balls shall be sufficient to reject a candidate. The Council shall have power to nominate active members.

Candidates for membership shall, at the discretion of the Council, present a paper to the Association showing clinical study of merit.

Section 3.—Any one who has been a member in good standing in this Association for twenty-five years is automatically transferred

* As amended May 10, 1916.

to life membership unless he expresses, in writing to the Council, his desire to remain an active member; and shall thereafter be exempt from all dues and duties, except the cost of Transactions of the Association, but shall retain all the privileges of membership.

Section 4.—Anyone who has been an active member of this Association for twenty (20) years in good standing, and has attained the age of sixty (60) years, may become an Emeritus member by expressing his desire to do so, in writing to the Council, and he shall thereafter be exempt from all dues and duties of the Association.

Section 5.—Any member of the ssociation absent from the meetings, in person or by contributed paper, for three (3) consecutive years, without sufficient cause, shall be dropped from the list of members by vote of the Council.

Article IV.—Officers.

Section 1.—The officers of this Association shall consist of a *President,* two *Vice-Presidents,* a *Secretary and Treasurer,* and a *Recorder,* who, with the Delegate to the Executive Committee of the Congress of American Physicians and Surgeons or his alternate and five other members, shall constitute the *Council* of the Association.

Section 2.—Nominations. The officers, including the Council, shall be nominated by a committee of five (5) members, which committee shall be nominated by the President at the first session of each annual meeting and shall report at the business meeting.

Section 3.—*Elections.* The election of officers shall take place at the business meeting. A majority of votes cast shall constitute an election.

Section 4.—The President, Vice-Presidents, Secretary and Treasurer, and Recorder shall enter upon their duties at the close of the annual meeting at which they are elected, and shall hold office until the close of the next annual meeting, or until their successors are elected.

Section 5.—Members of the Council, other than the President, Vice-Presidents, Secretary and Treasurer, shall hold office for five (5) years.

Section 6.—*Vacancies.* Any vacancy occurring among the officers of the Association during the year may be filled by the Council.

Article V.—Duties of Officers.

Presidents and Vice-Presidents.

Section 1.—The President and Vice-Presidents shall discharge the duties usually devolving upon such officers. The President shall be *ex-officio* Chairman of the Council.

Secretary and Treasurer.

Section 2.—As Secretary, he shall attend and keep a record of all the meetings of the Association and of the Council, of which latter he shall be *ex-officio Clerk*. At each annual meeting he shall announce the names of all who have ceased to be members since the last report. He shall notify candidates of their election to membership. He shall send a preliminary notification of the annual meeting two (2) months previous thereto, and the programme for the annual meeting at least two (2) weeks previous to its assembly, to all the members of the Association. He shall also send notification of the meetings of the Council to the members thereof. At each annual meeting of the Association he shall read the minutes of the previous meeting and of all the meetings of the Council that have been held during the current year.

Section 3.—As Treasurer, he shall receive all moneys due, and pay all debts therewith. He shall render an account thereof at the annual meeting, at which time an auditing committee shall be appointed to report.

Recorder.

Section 4.—The Recorder shall secure the papers read and also proper notes of the discussions thereon and shall superintendent, under the direction of the Council, the publication of the Transactions.

Article VI.—Council.

The Council shall meet as often as the interests of the Association may require.

Four (4) members shall constitute a quorum.

It shall have the management of the affairs of the Association, subject to the action of the Association at its annual meetings.

It shall consider the claims of candidates recommended to it for admission to membership.

It shall not have the power to make the Association liable for any debts exceeding in total one hundred dollars ($100), in the course of any one year, unless specially authorized by a vote of the Association.

It shall have the entire control of the publications of the Association, with the power to reject such papers or discussions as it may deem best.

It shall have power to nominate active members at the annual meeting.

The Council shall have power to invite any gentleman, not a member, to read a paper at the annual meeting, on any subject within the scope of the objects of this Association.

The Council shall determine questions by vote, or—if demanded —by ballot, the President having a casting vote.

The Council shall constitute a Board of Trial for all offences against the Constitution and By-Laws, or for unbecoming conduct, and shall have the sole power of moving the expulsion of any member.

The President, or any two members, may call a meeting, notice of which will be transmitted to every member two (2) weeks previous to the meeting.

Article VII.—Papers.

Section 1.—The titles of all papers to be read at any annual meeting shall be forwarded to the Secretary not later than one (1) month before the first day of the meeting, in order to appear on the printed programme.

Section 2.—No paper shall be read before the Association which has already been printed or been read before another body.

Article VIII.—Quorum.

A quorum for business purposes shall be ten (10) members.

Article IX.—Amendments.

This Constitution may be amended by a four-fifths (4/5) vote of all the members present at an annual meeting, provided that

notice of the proposed amendment has been printed in the notification of the meeting at which the vote is to be taken.

BY-LAWS.

(1) Meetings of the Association shall be held annually.

(2) The time and place of the meetings shall be determined by the Council.

(3) The dues of active members shall consist of an annual assessment not to exceed seven and a half ($7.50) dollars. Members in arrears shall not be entitled to vote. Those in arrears for two (2) years may be dropped from membership by recommendation of the Council.

(4) Order of business meeting.

First day:—

Calling the roll of members.
Minutes of previous meeting.
Secretary's and Treasurer's reports.
Recorder's report.
Report of Committee of Arrangements.
Appointment of auditing committee.
Appointment of nominating committee.
Report of Council on recommendations for membership.

Second day, Morning session:—

Reports of nominating committee and auditing committee.
Election of officers.
Election of members.
Nomination of candidates for membership.
Report of committee on health resorts.
Unfinished business.
Miscellaneous business.
Adjournment of business meeting.

PRESIDENT'S ADDRESS.

Delivered at the thirty-ninth annual meeting of the American Climatological and Clinical Association, held in Washington, D. C., May 2d, 3d and 4th, 1922.

Members of the American Climatological and Clinical Association:

You have made me President of the Association for this year,—a great honor, for which I thank you heartily.

May I first speak a little of the Association. That it is different from others, is delightfully evident among its members. That which makes it different from other medical organizations, and membership in it more prized, is the unexceptional and evident presence of mutual friendship. Such friendship is always valuable, helpful and inspiring. It creates a social atmosphere in which our annual meetings are passed cheerfully, satisfactorily and profitably. Every one seems to take an interest in the work and achievements of every other member. Such is the prevailing spirit of the dear old Climatological as it was originated and has continued through almost forty years of vigorous existence. This spirit and this sentiment well describe the little group of a dozen men who were our original members. Of these, three survive enjoying perfect clearness of mind and unabated desire to work, with remarkable ability to do so. As they continue to build their careers of work, well done, the world is thereby made happier, as is accredited to them by all hands. The standard was placed high and kept inviolate by our originators and those who have followed. All members must recognize that each one must continuously add to the work and purposes of the Association. Inaction is abnormal to our history.

The Association has helped to better equip physicians for their work—made them more efficient in combatting disease and thus has brought better health and happiness to mankind. When the correct history of medicine is written, among those holding first rank will appear enrolled, the names of many of our members. It is our

earnest hope and the past makes us expect that none of our older members in either years of life, or membership, shall consider retirement from active participation in all our work and activities. The hand of time has not dulled the ability of *any* member for active brilliant work. Appearing in nearly every volume of our transactions are to be found papers by men it is true of advanced years, whose continued vigorous mentality is made evident in these papers. I think it would be well before turning from this subject to name those original members who are happily still with us. They are Drs. J. Hilgard Tyndale, James C. Wilson and Beverley Robinson.

Then it would be well also to mention the names of those others who have been members and builders of our history for twenty-five years or more. They are Drs. Alton, Blackadder, Bridges, Coolidge, Ford, Gibson, Rice, Rogers, Fremont-Smith, Babcock, Boardman, Bonney, Bowditch, Sanger Brown, Buttler, Campbell, Daland, Coleman, Edson, Gardiner, Hance, Hart, Heffron, Jayne, Otis, Phillips, Platt, W. D. Robinson, H. Longstreet Taylor, J. Madison Taylor, H. F. Williams. The mention of these names is an inspiration, helpful, stimulating, for we associate in our thoughts how much medicine owes to their work, and so we are proud. We will expect and receive quite as much from our younger members and so your Association will increase in merited honor.

So long as we were a Climatological Association only, our field of activity as an organization was unduly limited.

Climate cannot be dissociated from other aids to healing. Recognizing this the name was changed by the addition of the word Clinical. All parts and organs of the body have a greater or less interdependence in the performance of their functions, therefore, by creating a greater vigor in the healthy organs, the sick ones are helped. To study the climate alone tended strongly to restrict study to the diseases in which it had been most used as a remedy, that was the diseases of the lungs. By the establishment of our broader field of study, we become more successful in treating ills and enfeebled condition associated with a principal disease, leaving the cure of the latter a much simpler and more promising procedure. As for example, the advantage gained in treating lung tuberculosis by having all other pathogenic germ activity and especially focal infec-

tions, eliminated as far as possible is manifest. The same is true of nearly all other pathogenic diseases which are the diseases in which climatic influences are most effective, and these are the diseases with which the Association has most to do. Now at our meetings the varied programs must bring to each member and especially him who is a medical specialist, information that will be of aid to him.

As stated our next meeting will celebrate our fortieth year of existence as a national medical organization. Might it be well for a committee of four to be appointed to review and comment on our work during these past forty years, forty-five minutes being allowed each member of the committee to present the review of his alloted decade, making one session of time in all. By such comparison with the past, the present would be more effectively and forcefully impressed.

Again, as there are now more deaths caused by cardiac diseases than by lung tuberculosis, and as a diseased heart so unfavorably effects the successful treatment of associated diseases, would it not be well to have one session given occasionally, principally, to the study of heart diseases, especially considering their relationship to and influence on other diseases.

I would recommend the establishment of another, a permanent committee to study and make annual report on advances and aids in medicine and allied or influencing sciences. The work of such a committee would be most instructive, and by citing references could point the way to securing most valuable information in great detail when members so desire. It would be the duty of each member of the Association to report to such a committee newly discovered evidence developed in their own work, and to call attention to articles met in reading, or facts learned in consultation or medical meetings. A capable alert committee keeping in touch with all sources of information, aided by the co-operation of our members, could surely present one of our most valuable annual contributions.

It seems to me a very unusually enjoyable and profitable meeting could and should be arranged for our next, our fortieth anniversary. The members could be stimulated to make it a real reunion of those always present, usually present, and some times present. It could be of such scientific and social interest as would in every way

most favorably impress the meetings following, leading on to a wonderful semi-centennial in 1932.

Our program is long but can easily be gotten through with by alert activity in our sessions. This will need co-operation of all. The official time of fifteen minutes for reading of papers must be adhered to. The points desired to be brought out in discussion should be concisely presented, but sufficient time allowed to every one who wishes to speak. Generous consideration for the time needed for the presentation of each paper and, discussion thereof should be kept in mind.

We now must turn to our program which gives generous promise of securing our heartiest commendation.

REPORT OF THE COMMITTEE ON CO-OPERATION WITH THE AMERICAN METEOROLOGICAL SOCIETY.

Dr. Hinsdale: During the year the members of this committee have discussed in person and by correspondence with the members of a similar committee of The American Meteorological Society the matters of common interest and the question of co-operation in research.

In April a joint meeting of the two committees was held at Hot Springs, Va. Throughout all the sessions a spirit of cordial co-operation and desire to be of mutual aid was manifest.

As the result of these conferences your committee make the following report and recommendations:

I. We believe that the present need in medical climatic research is along the line of a more accurate understanding of fundamental physiologic reactions to meteorologic and climatic components. In such study the technical knowledge of members of each society can be mutual benefit.

II. The publication of a joint volume or the institution of a concerted research does not appear desirable at the present time.

In this connection the question of publishing a revised edition of the Report on Health Resorts was carefully considered. It does not seem advisable just now for our Association to undertake the labor and expense of this work; and the Committee of the American Meteorological Society felt that the climatic and meteorological data for such volume was purely a matter of Weather Bureau statistics readily obtainable when wanted and not calling for creative co-operation by their Society.

III. For the present it would appear most useful for the advance of scientific work by members of either society on topics of common interest for them to have the means of easily availing themselves of the knowledge or experience of the members of the other society.

To secure this we make the following recommendations:

First: That this Committee on Conference with the American Meteorological Society be continued as a standing committee of our Association.

Second: That the Secretary of the American Climatological and Clinical Association keep a list of the members of the American Meteorological Society in order that our members may avail themselves of it for the useful exchange of information or queries; and

Third: if possible he obtain a check list of the members of the American Meteorological Society who would be interested to receive directly reprints of papers on climatological or physiological subjects read before our Association.

Fourth: We strongly urge every member of our Association to become an Active or an Associate member of the American Meteorological Society.

Carroll E. Edson,
Guy Hinsdale.

THE VALUE OF VENAESECTION.

By R. FORTESCUE FOX, M.D., M.R.C.P.
36 Devonshire Place, London, W.1.

Honorary Member, American Climatological and Clinical Association, President of the International Society of Medical Hydrology.

The present paper is intended to present, briefly, the results of my experience as a physician in the use of venaesection.

My attention was first directed to the immediate relief that sometimes follows blood-letting in acute cases when I was resident house physician at the London Hospital in the wards of the late Sir Andrew Clark. In Charlotte ward lay a woman, suffering from what some believed to be a mediastinal growth. Several times I was called at night to relieve her intense dyspnoea and pain, and at last, noticing the cyanosis, bled her, which was an easy matter, as the veins were much distended. The attack immediately ceased, and never recurred with the same violence. After that she was bled again from time to time, and the autopsy revealed an aortic aneurysm. I was a junior at that time, and remember that Sir Andrew publicly thanked me for re-introducing the practice of venaesection into the wards of the London Hospital.

From that time onwards I have employed this neglected remedy on many occasions, and have never observed any ill effects from it. Sometimes certainly it has done no good, sometimes the benefit was problematical, but in many cases—some of which I have followed for a long period of years—I have seen great relief from immediate symptoms, serious dangers have been averted, and chronic disorders have been controlled.

I have always taken blood with simple aseptic precautions from the most prominent vein in the bend of the elbow, and without pain, as a drop of weak solution of cocaine is first injected over the vein, and an ordinary lancet used which usually opens the vein with a single cut. Sometimes in emergencies I have used a sharp penknife. When the tourniquet is removed from the arm, dry lint and a band-

age are applied to the wound. I have usually recommended that the patient should remain recumbent for 48 hours after venaesection, in order that the circulation should remain as quiescent as possible. In cases of high arterial tension, if this is done, the pressure often settle down to a lower level in the course of a few days.

My observations, about seventy in number, fall into four groups, and may be classified as follows:—

(1) *To arrest cerebral haemorrhage.* The first case of this kind went for to satisfy my mind that bloodletting was a safe proceding, even in the aged.

Case I.

A lady nearly 70 years of age, stout and plethoric, suffering from indigestion, shortness of breath and discomfort in her head, came to a certain sulphur spa and drank the waters without advice. The congestive symptoms were dangerously increased, and one afternoon whilst sitting on the sofa she suddenly lost her speech, became confused, dropped the book she was reading, fell back and became unconscious. I saw her a few minutes later, and found the unconsciousness deepening into profound coma. The face was purple, the pupils fixed and dilated, the limbs moved convulsively at intervals, the right cheek flapped in respiration. I bled her immediately from the left arm. The force with which the stream came was extraordinary. Presently the convulsive movements ceased, and the pulse which had been very excited sank in frequency and force. Believing that no half measures could suffice to check the haemorrhage probably taking place in the brain, I determined to go on almost as long as the blood would flow. In the end I took 35 ounces. In eight hours she recovered consciousness, and on recovery declared that she was better than she had been for years. Her right leg and arm were weak for some months afterwards, and she was liable to confusion of speech and some want of memory for words when taxed with conversation. For several years afterwards, leading a guarded life, she remained quite well.

Venaesection has been successfully employed in cases of haemorrhage with fractured base of the skull. It has been supposed that bloodletting might be injurious if used for a hemiplegic seizure, due to thrombosis. It is easy to estimate the blood tension, both in the

arteries and veins. Where the tension is high, the veins full and the heart laboring, venaesection cannot fail to do good and probably favors the passage of blood in areas where it is impeded by venous pressure. It is remarkable how quickly it tranquillises the excited action of the heart. Why should reduction of venous pressure favor thrombosis?

Individuals with high blood tension are often first seen by the medical man at the onset of cerebral symptoms,—especially one-sided numbness and tingling, flushing, mental confusion and aphasia. I have observed that a prompt venaesection has given immediate relief in such cases, and removed all cerebral symptoms. Even after 24 hours, when the patient is hemiplegic and comatose, if the blood pressure is above normal, or the heart's action excited, or the veins in the neck at all prominent, 15 ounces or more of blood should be taken from an adult man. And if there are signs of improvement when the blood flows, it is well to continue until the pressure in the veins is reduced and the blood flows slowly.

It is generally recognized that there are not a few families in which the men, and even the women, are liable to die of "a stroke" or "apoplexy" between 60 and 65 years of age. The lesion seems to be always, or nearly always, a haemorrhage. Public and professional men appear to be particularly liable to this "accident,"—which befalls them after a long period of continuous brain work, accompanied by an insidious and perhaps unrecognized increase of blood pressure. Not a year passes but we suffer, as I conceive unnecessarily, the premature loss of medical men of about 60 years of age, at the height of their intellectual powers. For my part I am satisfied that we have in venaesection an unfailing remedy in these cases,—chiefly of course preventive, but even when the harm is done, sometimes, as in the case that I have quoted, followed by complete recovery. Many good observers in all countries are agreed as to the value in apoplexy. A timely venaesection would add many years to the lives of not a few of our greatest men: and it is lamentable that in spite of so much evidence it is still unemployed and neglected by the vast majority of medical practitioners.

(2) *Acute Spastic Conditions of the Circulation.* The case of *aneurysm* cited above is a good example of the seizures from acute

disturbances of the circulation, shown by pain and breathlessness, for which venaesection provides the best, and perhaps the only, relief. Another case of aneurysm is worth recording: also in a woman aged 34.

After a period of much physical and mental strain, she consulted me for swelling at the base of the neck, with much throbbing and distress. I found an aneurysm of the subclavian artery, possibly congenital, and Mr. (now Sir Charles) Ballance, who saw the case with me, feared that a rupture might be imminent. The patient had been already confined to bed and ice-bags applied. She was bled on many occasions, to 10-12 ounces, and expressed herself as delighted with the immediate relief, from quieting of the circulation. Fourteen years have elapsed, and she is quite well.

The same relief is often obtained by quite small venaesections in *angina pectoris,* the sedative effect being out of proportion to the quantity of blood removed.

Irregular spastic conditions of the circulation are an invariable feature of convulsive seizures from toxic or other disturbance of the higher nerve centers. *Epilepsy* (especially continuing fits, *status epilepticus*), puerperal *eclampsia* and *uraemia,* with dyspnoea or convulsions. In all these cases venaesection is indicated, and the mechanical aid to the circulation only partly explains the relief obtained.

(3) *Embarassed Heart.* In many acute illnesses—*mitral disease, bronchitis* with *emphysoema,* acute *congestion* or *oedema* of the lungs—the pulmonary circulation is dangerously impeded, and blood accumulates in the right side of the heart and superficial veins. This is shown by fulness of the veins of the neck and arms, epigastric pulsation, dusky blueness of the skin and dyspnoea. In spite of the poor and irregular pulse a venaesection to a moderate extent (6 to 10 ounces) is in my opinion the correct practice, tides over the danger, and may be repeated at intervals if the obstruction recurs. Here also the amount of blood removed seems out of proportion to the relief obtained.

(4) *Chronic Spastic Conditions of the Circulation.* A large proportion of my observations refer to men and women suffering from increased blood pressure, coming on usually in middle life

(sometimes earlier), and if not relieved, tending to get gradually more severe, and leading on to anginiform attacks and cerebral haemorrhage. In some the condition is obviously spastic and functional, due to excessive arterial tonus (angio-spasm) in some areas and relaxation in others, leading to an irregular distribution of the blood, for which one would not suppose that blood letting would be beneficial.

Case II.

A man, nearly 44 years of age, consulted me for sensations of heat and throbbing in the head, especially in the occipital region, and a sensation of burning in his face, increased by stooping, as in packing his portmanteau, and on going to bed, the throbbing causing troublesome insomnia. This had been getting very uncomfortable for three years, although since a boy he had always a tendency to "blood to the head." His mother died of "angina pectoria," his father of apoplexy; four brothers were "gouty," three sisters "rheumatic." He was a strong man, living in the fresh air, driving his own motor, taking much active exercise, but no stimulants. He married at 22, and was intemperate in sexual indulgence. His circulation was always peculiar, the hands and feet being cold in winter and hot in summer.

On bleeding to 10½ ounces I noticed that the venous pressure was very high, the arterial pressure being 135-140 before and 120 after the operation. My notes state "Vasomotor disorder (ataxia), peripheral and distal spasm (skin and extremities), atonic condition in occipital region and head generally." In one month the blood pressure fell below 100, and he remained for some time free of symptoms. Within the next seven years he returned to me on four occasions for venaesection, which always relieved his symptoms. Very similar observations have been made by Dr. W. F. Dutton and others.

Case III.

A lady aged 47, of gouty inheritance, who had had rheumatic fever at 29, suffered from a uterine polypus with frequent haemorrhages. The heart presented a systolic murmur with a highly accentuated second sound. She complained of occasional giddiness and dyspnoea and anginiform pain during the cold winds of winter. At

49 the polypus was removed and the haemorrhages ceased. There followed a sense of heat, flushing, precordial pain, depression, restlessness, gouty swellings and neuritis. Treatment at Aix-les-Bains relieved the high tension, as also in the following year prolonged baths at Loeche-les-Bains (130 hours in the baths). Two years later, the congestive symptoms having returned, I bled to 12 ounces with marked relief of all symptoms.

It is unnecessary to multiply examples. One of the most recent is a rather restless and neurotic patient whom I have watched for 25 years, and have had to bleed on several occasions for circulatory distress. Last year she had another access of high arterial pressure, at 76 years of age, concurrently with an attack of glaucoma. She was bled before the iridectomy. The circulatory excitement was at once allayed, and the blood pressure sank gradually (from 170 to 135 systolic), with relief to all symptoms.

In many cases of atheroma and arterial degeneration the pressure is liable to similar periodical and dangerous increase, apparently from phases of hypertonus or spasm of the superficial or other vascular areas. In these cases x-rays reveal an hypertrophied and labouring heart and perhaps dilatation of the arch of the aorta. The arteries accessible to palpation may be hard and cordy, partly from degeneration and partly from hypertonic spasm. If we find the pressure rising to a danger point with the usual symptoms (heat and throbbing in the head, etc.) I submit that venaesection ought to be promptly, and if necessary repeatedly, used, to 15 or 20 ounces or more. Such persons are often inclined to be stout, and show signs of congestion (hyperpiesis). I have never observed ill effects from venaesection, even when there was well-marked cardio-vascular degeneration. On the contrary venaesection eases the burden of the ventricle, not only directly and immediately, but by a remote and indirect action, as the careful observer will notice in the weeks and months that follow.

It is probable that the *rationale* of this treatment, both in acute and chronic disease, will be made clear not so much by experiments upon animals as by precise observation of the patients submitted to it. Data are needed on the effect of venaesection on the blood (immunization, viscosity, toxicity, etc.) in specific fevers, in pneumonia,

rheumatism, etc., following the very suggestive investigations of Shroeder and also upon arterial and venous pressures, including both the immediate and remote effects. Those who have had long experience in practice must have seen many persons who are liable to periodical increases of arterial tension—related to the season of the year, and also related to the time of life. We must recognize that there are natural methods for relieving over-tension, which are often supplemented by epistaxis and other haemorrhages: and that the middle period of life and after is a period of danger for pressure maladies. We have now ample means for estimating and recording the life-curve of the pressure of the blood from year to year, and for estimating the point of danger. I suggest that continuous "barometric" records should be kept for all of our patients who show signs of upward deviation from the normal curves, and that venaesection should not be deferred until the danger point has been reached.

AUTOMOBILE EXHAUST GAS AS A HEALTH HAZARD.

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With every advance in industry and the arts there is liable to be associated some new physiological strain or health hazard. This has been true of machinery, of the chemical industries, of electricity, and it is now proving true of man's latest and most widely used utility, the automobile. As a rule, however, the hazard has been confined to some particular group of workers or class of persons. The risks involved with the automobile are, however, almost universal. The loss of life in accidents as reported in the daily papers is so common that it is receiving serious attention although not yet very effective measures of amelioration,—as every one knows.

But it is not very gradually realized that the exhaust gas from automobiles constitutes scarcely a less serious health hazard. Fortunately, engineers are beginning to recognize that the sanitary side of large public undertakings and even of some private developments deserves study and the public is learning to expect this protection. Accordingly when plans were made for the construction of the first long vehicular tunnel in this country, these plans included extensive investigations of the basic facts necessary to the ventilation of such a tunnel so as to render it not only safe but practically innocuous to its passengers. This is the more important as such tunnels will probably to a considerable extent replace bridges in future both because of their cheaper construction and because they do not obstruct the water ways.

The Commission of the states of New York and New Jersey charged with the construction of the vehicular tunnel, or rather tunnels, under the Hudson River arranged with the United States Bureau of Mines to undertake these basic investigations. The work was divided into several special problems of which we need here only consider two, namely: (1) the amount and chemical composition of the exhaust gas produced by various cars under various conditions

of speed, load, and temperature; and (2) the physiological effects of the substances in exhaust gas and their allowable amounts not involving appreciable ill effects when such gases are breathed.

The first of these problems was placed in the charge of Mr. A. C. Fieldner at the Bureau of Mines Experiment Station in Pittsburgh. An extensive investigation was carried out by him and his collaborators and has shown that to a large extent the deleterious character of exhaust gas of automobiles arises from the tendency of drivers to adjust carbureters so as to afford a maximum of power rather than to utilize fuel with the maximum efficiency. (1) The gasoline mixtures used are generally much richer than would be best both from an engineering and sanitary standpoint. More flexible carbureters and greater use of the adjustments so as to attain the required speed and power with a minimum of fuel would only result in reducing the waste of gasoline but in an exhaust gas distinctly less injurious to health. These investigations show that approximately one-third of the fuel value of gasoline is wasted and that the carbon monoxid, the particular toxic constituent of exhaust gas, averages about seven percent. As regards the total quantity of exhaust gas produced, it is sufficient to say that most passenger cars with the usual carbureter adjustment produce from one to two cubic feet of carbon monoxid per minute.

The second problem above referred to was investigated by the writer, with the collaboration of Dr. H. W. Haggard, Dr. A. L. Prince, M. C. Teague, R. M. Wunderlich, and a number of chemists for the analytical work, in the laboratory of Applied Physiology at Yale University. (2, 3). Our investigations have completely confirmed the conception of Paul Bert Haldane and other investigators that carbon monoxid does not form a permanent compound with the haemoglobin of the blood but competes with oxygen. It has an affinity about 300 times as great; it is able not only to displace oxygen but to be again displaced by oxygen, when a person who has absorbed a certain amount of gas breathes pure air, or air enriched with oxygen. Our investigations also show that carbon monoxid has no other toxic characteristic except its combination with haemoglobin and exclusion of oxygen; it has no direct effect upon nerve cells; all of the nerve degenerations induced by carbon monoxid are due to

this asphyxia. Dr. Haggard in this laboratory has indeed even succeeded in growing nerve tissue in considerable amounts of carbon monoxid. (4). Animals such as flies and other insects which have no haemoglobin are as entirely immune to any effect of carbon monoxid as they would be to nitrogen, the most inert of gases.

Our investigations show further that when gasoline distilled from petroleum is used, practically the only toxic constituent in the exhaust gas is carbon monoxid. When, however, benzol and probably other related substances are present, as in some of the automobile fuels now sold, the amount of these substances escaping in the exhaust gas adds decidedly to its toxicity. (5).

Our investigations were carried out at first on ourselves and other members of the investigating staff in an air tight room lined with sheet iron. The data so obtained were later confirmed by large scale experiments in which a dozen or twenty people at a time sat or moved about in a garage building in which a Ford car was installed. A drop of blood was drawn from a finger and analyzed colorimetrically for the percentage saturation with carbon monoxid; the pulse was counted; the volume of respiration measured; and the effect of exercise, after running up and down four flights of stairs, was noted. The Romberg test for steadiness was used in some cases.

The best of all indications proved, however, to be the headache induced by carbon monoxid. This is the characteristic oxygen deficiency phenomenon observed in persons at great altitudes and induced in nearly all persons by certain degrees of saturation with carbon monoxid although there are some individual differences in the exact point at which it appears. It is usually of the throbbing frontal type, more rarely occipital. It easily passes into nausea and is accentuated by exertion.

Space forbids a detailed description of theory or experiments, but data was obtained sufficient to prove that the rate at which carbon monoxid is absorbed from air is such that at least an hour is required for the blood to attain a degree of saturation equal to half that which would finally be attained by a stay of indefinite duration.

From these demonstrations the following rule was derived for practical use: When the time is expressed in hours and the saturation in parts per 10,000, there will be no appreciable physiological

effect when the product of the time multiplied by the saturation equals 3; when the product of time by concentration equals 6, there will be in some persons slight effect; when it equals 9, there will be in nearly every one a distinct effect; when it equals 15, the conditions is dangerous. A concentration of carbon monoxid not exceeding 4 parts in 10,000 is therefore recommended as a practical standard for vehicular tunnels.

It is quite certain, however, that this standard is very often exceeded in garages. Severe headaches are extremely common among mechanics and others who work around automobiles; so much so that repeated partial asphyxia is a distinct health hazard.

The data given above will serve to indicate the character of the hazard in the small private garage. During the winter months in the northern states asphyxiations in such private garages are unfortunately quite common. The owner starts his car before opening the doors in order to get the engine warm. If the garage is 10 x 10 x 20 feet, giving a capacity of 2,000 cu. ft. and the car produces 1 cu. ft. of carbon monoxid per minute it requires only one minute for the saturation which we have recommended for the tunnel to be reached and in five minutes the atmosphere contains 20 parts of carbon monoxid in 10,000 of air, an amount sufficient to induce rapid asphyxiation. And of course if the car continues to run, the saturation mounts steadily toward a rapidly fatal concentration of carbon monoxid.

In addition to our experiments on men, which could not be pushed beyond the point of severe headache, we have performed an extensive series of experiments on animals which we carried to the point of extreme if not fatal asphyxiation. These experiments were performed with pure CO, with exhaust gas from automobiles, and with illuminating gas. They show that CO is practically the sole toxic constituent in exhaust gas when pure gasoline is burned. Illuminating gas, however, owes perhaps 20 to 25 percent of its toxicity to some other constituent, perhaps benzol. Gasoline, as sold in some places, however, is mixed with coal distillate which contains benzol and produces a correspondingly more toxic exhaust gas.

Gasoline vapor itself has been found in our experiments to have qualities somewhat like those of ethyl ether. (6). It is an anaesthetic but the stage of excitement passes through a very narrow region of

full anaesthesia into convulsions ended by death. (7). In the toxicity of exhaust gas unburned gasoline is not an appreciable factor. Workmen are, however, sometimes overcome when they go into a large tank in which gasoline has been stored. Benzene (benzol) is even more toxic as Dr. Alice Hamilton has recently emphasized. (8).

Finally these investigations have lead on to the study of the treatment of CO poisoning. Obviously the first step in any treatment must be the restoration of spontaneous breathing if it is stopped. For this purpose the manual prone pressure method of artificial respiration is to be recommended above any apparatus, for it is as effective, or even more effective, than apparatus, and it can be applied instantly, while the bringing and adjustment of apparatus involves delay. A delay of even a couple of minutes may sometimes be fatal.

The next step after spontaneous breathing must obviously be to accelerate the elimination of carbon monoxid from the blood. For this purpose the inhalation of oxygen has long been recognized as the specific procedure. (9). But the difficulty arises that after a long exposure to an atmosphere containing carbon monoxid, respiration is very much depressed and the inhalation of oxygen has no very stimulating effect upon the volume of breathing. It occurred to us to utilize the stimulating action of carbon dioxid. In a series of experiments on dogs we found that in animal which were brought almost to the point of death and then allowed to recover spontaneously in air, the period of elimination of carbon monoxid from the blood covered several hours. When the subjects were made to inhale oxygen the rate of elimination was at first not very much more rapid, the reason being that the oxygen was inadequately inhaled. When, however, a small amount of carbon dioxid was added to the oxygen a very active breathing was induced, by which the lungs were so effectively ventilated and the oxygen was given so free an opportunity to act upon the blood, that after a period of a quarter to half an hour nearly all of the carbon monoxid had been eliminated from the blood.

Recently the American Gas Association, representing practically all of the illuminating gas interests of the country, has requested Dr. C. K. Drinker of the Harvard Medical School to organize a commission for the further investigation of the subject of resuscita-

tion from illuminating gas poisoning. The members serving on this commission are Dr. Drinker, as chairman, Drs. D. L. Edsall, W. B. Cannon, L. J. Henderson; Mr. C. B. Scott of the Bureau of Safety; Dr. R. R. Sayers of the U. S. Bureau of Mines; Drs. Howard W. Haggard and Yandell Henderson. The last two named have been appointed a sub-committtee to investigate the efficacy of the above mentioned treatment. Through the courtesy and co-operation of the Consolidated Gas Co. of New York City, which has placed at our service its emergency wagons, and with the support of Health Commissioner Copeland, and the hospital authorities of New York City, we have been able to give the method an extensive test. The results indicate that a deeply asphyxiated patient who can be reached within a half-hour—and the utmost speed at this time is essential for success—and who is then given inhalations of oxygen containing 5 percent carbon dioxid, can be practically freed from carbon monoxid within 30 to 40 minutes. Frequently also consciousness returns.

The demand for cheaper gas on the part of the public and the rise in the cost of crude petroleum, the introduction of the mantle burner, the increased use of gas for cooking and heating purposes have inevitably resulted in an increased number of fatalities from illuminating gas asphyxiation. For those cases which can be reached in time the treatment above indicated will, it is our belief, not only increase the chances or survival, but also the probability of complete recovery and freedom from the distressing sequelae sometimes associated with gas poisoning.

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THE CLIMATIC TREATMENT OF TUBERCULOSIS.

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The psychology of the world has changed so rapidly in the last few years that one is completely overwhelmed in an effort to understand or analyze the factors that have produced this upheaval in the world's affairs.

Medical thought, surgical principles, therapeutic practice, medical education, have been revolutionized in the last decade for better or worse.

The newer theraputic method or discovery, the latest invention in instruments, the fad, fancy and fashion of some brilliant dreamer does not always prove the winning success that its spectacular literature would lead us to believe.

Those of us who have lived a quarter of a century in the practice of medicine know the storms we have encountered. We have been criticized and abused and when we landed we learned to know the land of health and happiness is pierced by a middle road that we now delight to travel. Competition in ideas is keen, often unjust. Hypercriticism is fashionable and the unkind restless nervous spirit of war is among the people.

We must realize that the modern patient is impatient, unstable, and changeable. The doctor of today is not his of tomorrow. The law of obligation, the mere matter of appointments at the office or for operation are more often broken. The patient who leaves for Silver City lands in California. Advice is unheeded or the last is the best. Appreciation of professional service has diminished and the wise counsel of experience is turned aside for the fad of the osteopath. The tubercular gland and joint are rubbed and the patient is content.

This is the age of the aeroplane, the automobile, the transcontinental flyer, the ocean grey-hound, the auto bandit, and the movie. This is the mechanical and amusement stage of the world. More than 10,000,000 men have been moving to or from the great noisy

guns of war. This is the age of vibration and storm, the quick lunch, the short sleep, short skirt and short hair. A gentle question may receive a burst of anger. A simple request to perform some simple duty may be met with the ultimatum to get some one else. The public and the profession will not wait.

With a propaganda against climatic treatment is it strange that many needy cases refuse to accept this great therapeutic aid. Is it peculiar that unthoughtful or mercenary physicians keep all cases at home?

It is true that many innovations, discoveries, and scientific publications are not progressive or practical but rather the product of an anti-school of thought which although destructive leaves no constructive or better plan of method in operation.

After many years of stupendous work, scientific research, volumes of literature, wonderful leaders in medical thought and organization, we are today without a specific for this disease.

During these two decades medical opinion and organization, not facts, have launched such an anti-climatic howl and propaganda that thousands of cases have been deprived of these additional benefits to arrest and cure, proper climate for the proper case affords. Opinion and general statements unsupported by facts are not only valueless but very dangerous in medicine. The genuine test of therapeutics in tuberculosis is not what you and I may think or believe, but the test of time and results ten years after treatment. Is the patient alive or dead? Are tubercle bacilli remaining in the sputum? Are rales persistent? Is there activity?

I believe that the therapeutic indications for climatic treatment of tuberculosis are as definite, important, and limited as in any other disease. There is no patient given more or worse advice, yet few are so helpful. The tuberculosis societies, boards of health, and public press have been so extravagant with instruction and information that the laiety feel that this is a disease they know how to treat and every patient become public property of the laiety.

No disease is more dependant on a plan of treatment for results. Success is in relation to the detailed management and study of the individual. Is there anything more pathetic than the migrating advanced lunger who goes West, sent by the family physician, to

camp or to board, to drink ditch water and worse, food from some distant town to cook himself, disheartened in a few weeks, that the great climate has not cured him, he lands in the County House with a hemorrhage or a terminal pneumonia and telegraphs his friends for money. As the evils of the saloon have produced the evils of prohibition so the virtues of climate are lost to many through faulty over-use by a careless profession.

It has been said that there is no such thing as an ideal climate. As a general statement regarding climate alone, that statement is true, but we believe that as far as the treatment of tuberculosis is concerned, there are a great many places in this country and abroad which afford an ideal climate.

Such a climate is high, dry and cool. It is the combination of these three factors which make up the ideal climate for the tuberculous. With particular reference to the Southwestern United States, there should be in addition, freedom from dust, storms, and mosquitoes.

The very grave mistake has been made of exploiting the whole dry region of the United States as a health resort, which is absolutely not the case. An ideal health resort must be favorable at all times of the year. In the Southwest, we have nearly all possible varieties of climate except wetness. Dryness is the only factor held in common. There are places where the summers are so hot that one could almost cook an egg on the pavement, and others with a summer so delightful that words fail to portray the real truth. There are places so cold in winter that one might as well be in the Adirondacks or Canada, and places so modified by latitude, altitude and dryness as to be relatively warm in winter and others which have the winter climate of Egypt. There are places where there are frightful dust-storms for four or five months of the year. Such are totally unfitted to be health resorts. The well known health resorts of the West, as viewed from a Detroit standpoint, are Colorado Springs, Las Vegas, Santa Fé, Fort Bayard, Fort Stanton and Silver City, New Mexico and Prescott, Phœnix and Tucson, Arizona. For perfectly apparent reasons we do not mention the large cities as such are not ideal places for consumptives, regardless of climate. We will never utilize our western climates to the best advantage until more brains are applied

to the question where to go. We have vast areas perfectly suitable for consumptives such as the Gila and White Mountain Forest Reservations but at present they contain none of the other essentials of proper treatment of tuberculosis; in other words they are wild and uninhabited.

In the belief that there is a comparatively ideal climate we offer our results. We have no doubt that similar results are obtained at institutions having equally or desirable climatic environment.

In Table 7 is exhibited the results obtained at the Trudeau Sanatorium. In these tables, he who runs may read the definite reply to the anti-climatic teaching of the past twenty years.

A critic may object that classification of tuberculosis is so difficult that even a perfectly honest intent, comparison of results in different institutions is unfair. To which we reply—"Very well, let us abolish the classification and consider only those who are discharged better or worse. Surely any one can tell whether a consumptive is better or worse."

What is the result? We offer Table No. 8 which exhibits the good and bad results at the Trudeau Sanatorium, The New Mexico Cottage Sanatorium and at Fort Bayard under the Army. They include altogether nearly thirteen thousand cases. In each and any class and taken together without classification, the results at the two latter institutions excel the former and the worse the class the better the results in comparison with those at Trudeau. In other words the sicker they are the more they need what the Southwest has to give them.

It is evident that 24% of cases are not amenable to climatic methods of treatment and we feel that such cases are best treated at home, in fact they are the type of cases that have instigated the anti-climatic propaganda.

The large number of advanced cases that come to Silver City speaks for itself. They are the failures of elsewhere. But even so they represent but the least number when one remembers that finances are such essentials in reaching this country.

There is a widespread existing belief too generally accepted and taught that a sanatorium patient from the West relapses quickly on return to his occupation at his home; that an arrest or so-called cure

made at home is more permanent. This is a statement to be challenged as to conscientious accuracy. The resulting relapse should truly be charged to improper action or treatment upon a patient's return to his surroundings and failure of local conditions to hold such patients at an adequate state of health.

Bullock says failure to hold a patient at the requisite good health is the rarest sort of happening when they remain under surveilance in this climate. In fact there are numerous patients who have taken up their residence here who in time have become actual cures under conditions of unusual stress under which they have repeatedly failed at home. Whatever this may be due to, be it climate or care, it is to be noted that results from Fort Bayard records are equal to ours and mostly better than any other group of records of which we have been able to obtain a copy. This is true of institutions richly endowed and long established, with medical staffs that merit the highest esteem. That climate has a vital influence on the end result is to us and others a proven fact. If fresh air is a factor in the result of tuberculosis treatment, then the fact that patients can, in entire comfort, get more of it than any other place in the mountains of the southwestern country should be an argument in itself.

That the Southwest has been maligned is true, it will continue to be, and this without exhaustive investigation, if any investigation at all. Certainly it has been in our presence disparaged by men concentrating their efforts on tuberculosis in some of the large cities who, to my certain knowledge, have never been west of Missouri.

Climatic treatment needs no argument, only facts and one statement. How many physicians and their families are found in home sanatoria? Practically none and yet the Southwest is full of them because they know that climate offers an additional chance worth while.

Climatic treatment is for the careful selected case and considered.

First—He must have more than twelve hundred dollars cash.

Second—He should be referred to some reliable physician or sanatorium.

Third—Good food and suitable sleeping accommodations are essential to success.

Fourth—The treatment of the patient and family conditions should be given careful consideration.

Conclusions.

1. Patients should be financially prepared for nine months or a year in a sanatorium.

2. No case should be sent away without reference to qualified medical supervision.

3. Statistics prove that early cases show 91—64% recovery, 60—42% moderately advanced and 10—34% far advanced varying with financial condition and temperament.

4. Cool, dry, high altitude with the greatest number of sunny days adds much to the percentage of recovery.

5. The selection of the climate, for the individual is of great importance.

DISCUSSION.

The President: We will now ask for discussion.

A Member: I want to say that I think this is a very valuable contribution, as I think there is a great deal to the climatic treatment of tuberculosis. Some cases cannot be treated at home under the best of conditions of finance and mental state. It requires climate in a great many cases, and I think we get better results by proper climatic treatment. I will not boost anyone's climate more than the other, but I think the conditions of climate, the dryness and coldness and elevation, are of the greatest importance, but I do feel we ought to go on record as a Climatological Association as being highly in favor of climatic treatment of tuberculosis.

The President: Is there further discussion?

Dr. Swan: I think any person, with a reasonable sense of modesty, who lives in a health resort, hesitates to say much about the advantage of climate, because he is open to the suspicion of having an axe to grind, but I think one point should be made that when you speak of climatic treatment of tuberculosis, it is not climatic treatment; it is the use of climate as one of the aids, one of the factors in the treatment. A patient going to the climatic resort for tuberculosis needs just as good care and supervision, and every other facility for an improvement as he does when he stays at home.

I was glad to hear what Dr. Shurly said as to relapses. I do not think there is anything in the general opinion about a person getting well in a high latitude going back to his original home at sea level, is much more likely to

relapse than if he had obtained a cure in his own climate. That, I believe, is absolutely not so. I know there are many cases that have become arrested at high altitude, and they have gone home and lived for years under the former conditions, and I am sure the statement is not so.

I think one other thing is important, the fact that in health resorts a good way from home there are comparatively very many less of incipient than advanced cases. I should say that the majority of cases that we see in Colorado have tried this thing out at home, or have delayed so long that they are not incipient cases. We do not get as good results as we would if they had come earlier, but there are a good many of them who would improve at home, but who do improve there.

How much is climate, and how much is the matter of living out doors under more favorable conditions is important.

For instance, we play golf and are happy in it, and I think a person is most happy playing the game, sitting outdoors in surroundings where there is sunshine and cheerfulness, than sitting out where it is gloomy and rainy.

THE PRESIDENT: Dr. Minor, will you discuss this subject?

DR. MINOR: I did not hear the paper, and I would be an extremely rash man to discuss this subject in a few remarks, because this is a very enormous subject.

What I can say is that I am satisfied that climate is valuable. If I had the disease and could possibly afford to have proper climate, with proper care also, I would consider myself extremely foolish to forego it.

The trouble is, so far as I am concerned, I find that metereology and climatology have advanced so little or at least my knowledge of it is so little, and I do not find many men who understand what is the thing that works. That something works, however, I believe. That one climate is better than another I am convinced as I can be of anything, and I think twenty-eight years' experience justifies me in forming such an opinion.

I think the experience of the world shows climate to be a valuable thing, but we have come to an era when some say you can treat a case in New York as well as in some good climate. I think that is an error. After all, in large, the truth is always the middle between the two extremes. We are apt to forget—I can say that to me there is no question but what climate is a valuable factor.

I do not want anyone to say that climate is everything, and forget the other important things, nor do I want to think it is of no value at all.

I believe my patients get well not only from any care I may be able to give them, but in those changes of climate they also get.

THE PRESIDENT: Is there any discussion?

DR. FORSTER: There are a great many men today who think that climate plays an important part in the recovery of patients, and there are a lot who do not, but the opinion that climatic treatment is of vast importance will finally be settled among us. There is a group of men who have climatic advantages in handling their cases. I went to Saranac Lake myself, and I went there because I heard of Dr. Trudeau, and because to my mind, as a patient, it was of the

utmost importance for me to get the best medical advice I could, and I feel I got it, but I feel also that climate was an element in the success of my recovery. I feel that the climate at Saranac was a distinct element in that case. If it is not, these gentlemen at Saranac and those who come from the west, and those of us in other climatic resorts, have no right to remain there. Instead of making the mountain come to Mohammed, I should think Mohammed should go to the mountain. There is no value in having our patients take long railroad journeys and make them go to extra expense; we should conduct our work at more convenient points and get the benefit of the experience and training we have to such centers where patients are a large congregation, and where we can do our work with greater facility.

Dr. Shurly: I have nothing further to add except it sems to me that the great point to this whole meeting is that this wave of anti-climatic treatment which is so prevalent through the middle west, shall have its limitation, and that the pendulum will swing back where it belongs, and giving the same identical sanatarium treatment, plus the additional value of climate, it has been demonstrated that fifteen per cent additional value in recovery is made. This will have to be thoroughly understood, and these statistics absolutely proved to be of very great value.

TEMPERATURE AND HUMIDITY FEATURES OF A NEAR-DESERT CLIMATE IN SOUTHERN CALIFORNIA.

By FORD ASHMAN CARPENTER, Sc.D., LL.D.,

LOS ANGELES.

Californians are sometimes charged with being fond of the superlative, but in the matter of highest and lowest temperatures, at least, the Golden State holds the record for American extremes. In 1913 the highest temperatures, 134 degrees (Fahr.) was registered on July 10, by a standard thermometer exposed in a latticed instrument shelter five feet from the ground in Death Valley. A little over two hundred miles southwest of that Valley and about ten miles above the ground (over Catalina Island) a standard registering thermometer recorded a temperature of 90 degrees (Fahr.) below zero. As this article has to do with desert temperatures and humidities, we will concern ourselves with the former phenomena and dismiss the latter with the statement that the sub-zero temperature was obtained by aerial soundings from a balloon.

Not only is 134°, Fahr., the highest official temperature in California but it is the world's record for maximum air temperatures. Death Valley in Southern California is the most perfect example of a desert of which meteorologists have records. A glance at the bas-relief map accompanying this article (Fig. 1) shows that this valley, in addition to being nearly 300 feet below sea level, is in the lee of the towering Sierra Nevadan Mountains. In addition, several inferior mountain ranges also intervene. Bearing in mind that the general circulation of the air in these latitudes is from the west, the aridity and accompanying high temperatures of Death Valley are readily explained.

As to living conditions in Death Valley the physical hardships encountered there may be imagined from a perusal of the records of nine years which show that the average July temperature is 101° and the average daily maximum is 116°. The late General Funston, when a young botanist, made some interesting meteorological

observations for the Government in the summer of 1891 in that region.* During the past decade, public spirited men have continued a highly interesting series of weather observations in that extraordinary country. Two years is the average length of life for a white man in Death Valley although the native Indians eke out their three-score and ten.

FIGURE 1.
Bas-Relief Map of Southern California.

Generally speaking, all the deserts of the world are caused by the same two conditions—they are in the lee of dessictaing winds and away from the general storm tracks. In order to briefly study these conditions, temperature and rainfall will be considered separately.

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* "Notes on the Climate and Meteorology of Death Valley, California," by Mark W. Harrington, Chief of Weather Bureau, Bulletin No. 1, 1892.

From the records of the Weather Bureau and Bartholomew's Physical Atlas, Vol. III, Meterology, (Fig. 2) it will be observed that Death Valley has the highest July mean temperature (over 100°) much higher than any data to be obtained from Tibet, Persia, Arabia, or Egypt. This chart also shows that the little city of Riverside in southern California has the unique distinction of having the lowest temperature (75°) and a January temperature only inferior in height to Yuma, Needles, or Death Valley in the United States. or to Arabia and Egypt elsewhere.

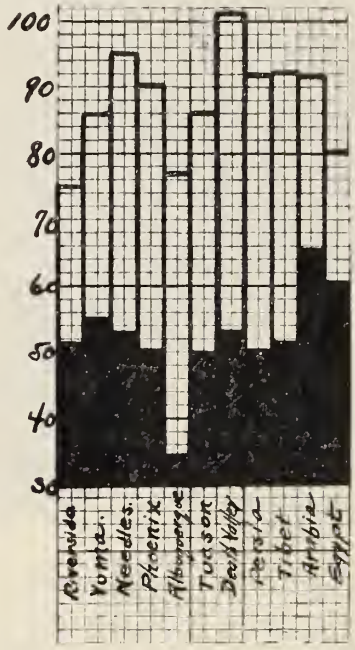

FIGURE 2.

Normal Mean Temperatures January and July; Riverside, California, Compared with Desert Regions of the Northern Hemisphere.

(July indicated by columns)
(January by black portions)

Data from U. S. Weather Bureau records and Bartholomew's Climatological Atlas. (Physical Atlas, Vol. III).

In the matter of rainfall (Fig. 3) Riverside has the greatest rainfall in January and the least in July of any of the regions named. This is caused by the proximity of this city to the coast; the Pacific Ocean being but 70 miles westward.

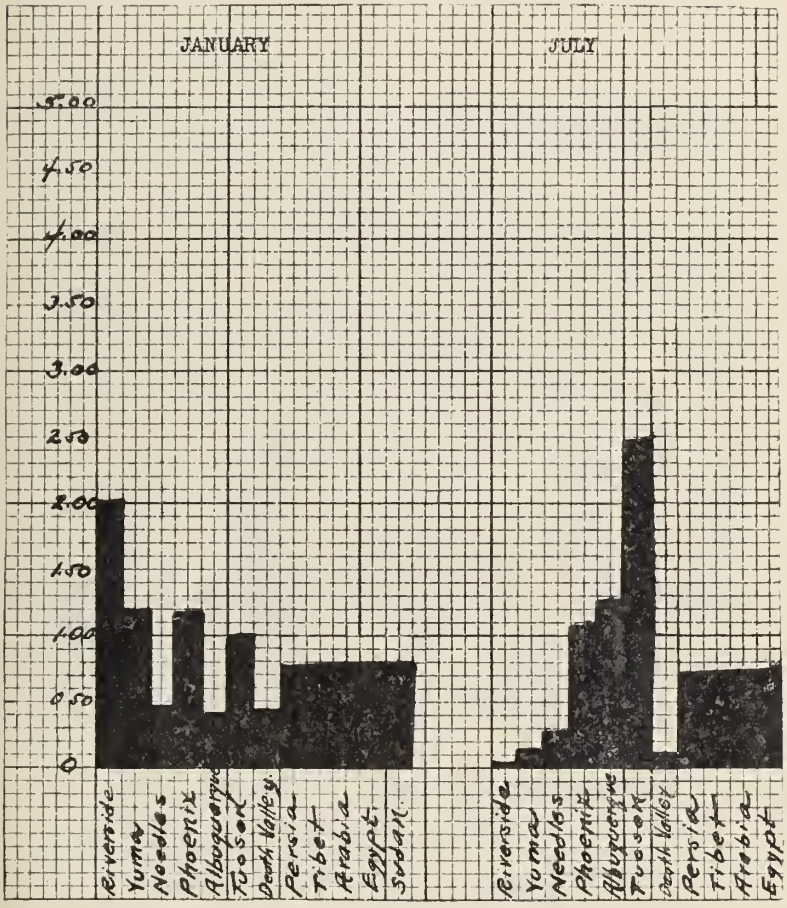

FIGURE 3.

Average Precipitation January and July at Riverside, California, Compared with Desert Regions of the Northern Hemisphere.

Data from U. S. Weather Bureau records and Bartholomew's Climatological Atlas. (Physical Atlas, Vol. III).

The distribution of temperature and rain throughout the year at Riverside is shown in Fig. 4. From April to November the thermometer rises to 90° or above, and from October to April it drops to 32° or somewhat lower. The rainfall records show the effect of the regular North Pacific storms in that February has the greatest rainfall.

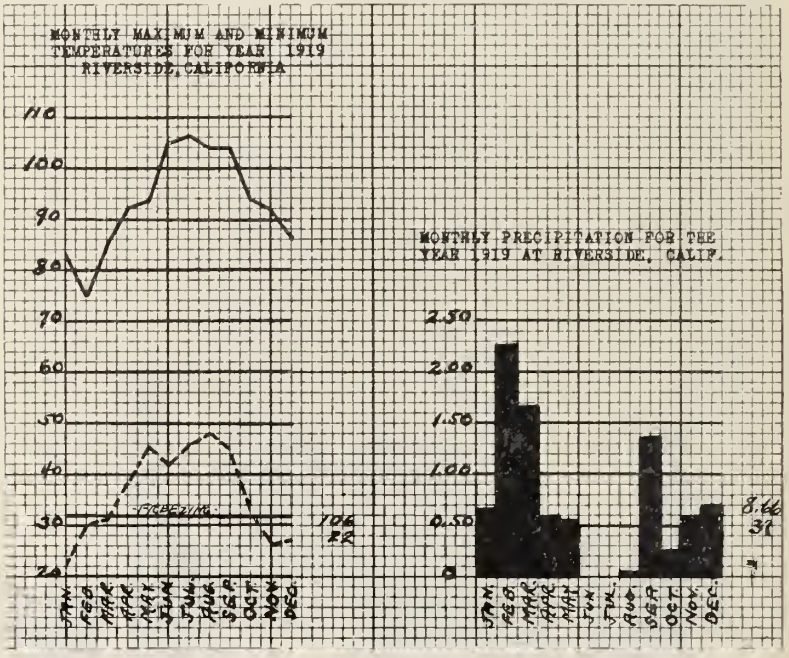

FIGURE 4.
Temperature Extremes and Rainfall at Riverside, California.

Data from U. S. Weather Bureau records.

Figs. 5 and 6 illustrate average hourly temperature in degrees and relative humidity in percentages of saturation for January and July. To what use can the physician put such charts? Selecting a definite relative humidity value which experience has shown to be favorable to a patient (60% for example), he can stipulate that between 6 p.m. of one day to 6 a.m. of the next, that the patient

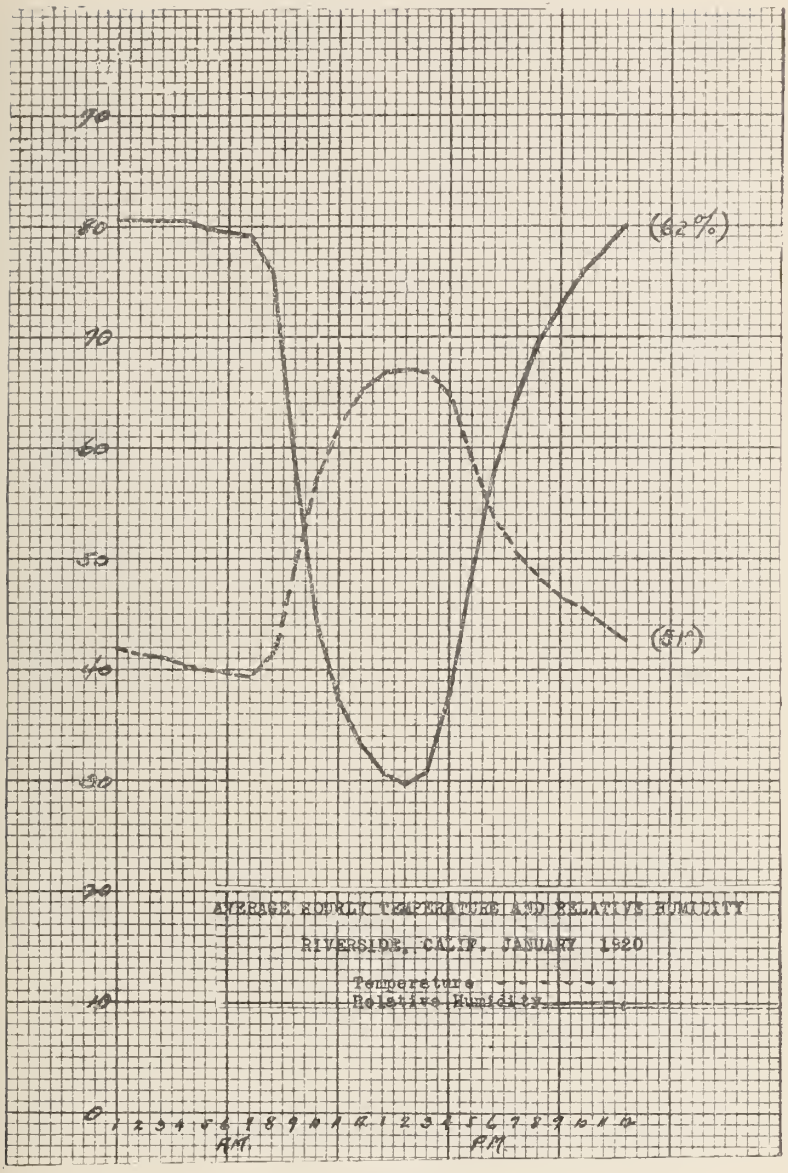

FIGURE 5.
Average Hourly Temperature and Relative Humidity, Riverside, Calif.
January 1920.
Temperature ---------- Relative Humidity ————
Data from U. S. Weather Bureau records.

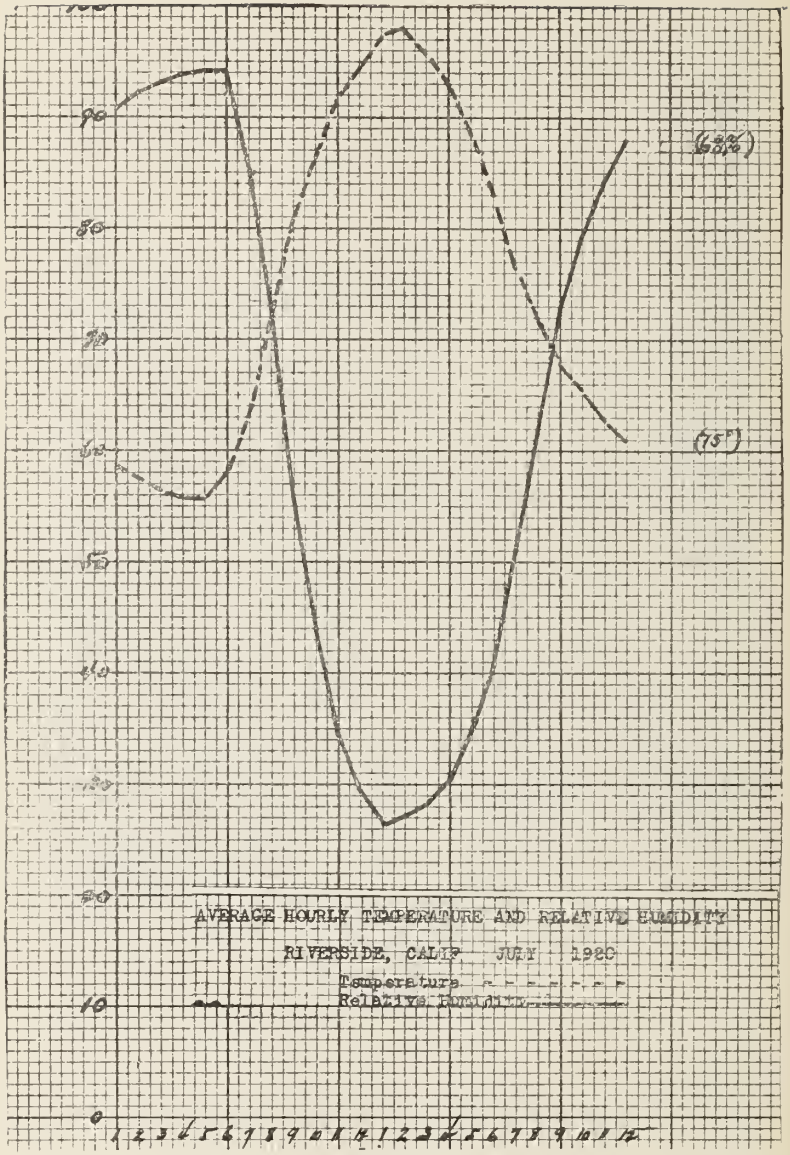

FIGURE 6.
Average Hourly Temperature and Relative Humidity, Riverside, Calif. July 1920.
Temperature ---------- Relative Humidity ————
Data from U. S. Weather Bureau records.

remain within doors where the air has ordinarily that percentage of moisture or less.

It would be useless to prescribe residence in a region unless the good and bad qualities of that climate be reckoned with. No place is free from disagreeable days of unpleasant weather; it is only when the peculiar advantages outweigh the disadvantages, that residence there becomes an aid to health. Local environment plays a most important part. For example, the exposure of the building or room to the sun and wind; the elevation of that room above the ground; and the height of and distance of neighboring dwellings are all important factors. Proximity to distributing points for meat and vegetable supplies, as well as easy access to centers of population for recreation and study—all play a most important part in regaining health or keeping it after once secured. There is one caution physicians should not overlook; the patient who may be sent to a place for recovery should be encouraged to make that region his permanent home. Too often the patient leaves a region where he has prospered physically to a region less favored climatically only to find a gradual diminution in healthy vigor which almost invariably ends in disaster.

CO-OPERATIVE RESEARCH ON THE ATMOSPHERE AND MAN.

By Dr. ELLSWORTH HUNTINGTON

Yale University.

I wrote a book on climatic changes, and I used a mathematical method and a geological method, and I gave the book to two friends at Yale, and asked them to read it. The mathematician read it. He said, "Well, that geological chapter is pretty theoretical." I said, "What about the other chapter?" He said, "I have nothing to say, because they are facts." I gave the book to a geologist. He said, "It is a fine, but I do not think much of the mathematical chapter."

There are several different methods of studying, and it is very interesting to me to observe this audience today. You are talking about the case method, the case, case, case, and it is a very valuable way. You are not so strong on mathematics. You have ten papers scheduled in two hours, and give half an hour to a paper. There are three minutes allowed to speak about it. One is the case method which you are using, another the laboratory method of experiments, and a third the statistical method. That system is climatological, as well as clinical, I understand, but still I see another part of the program that you have the climate in it.

A good many of us think that the climate is very important, and so important that the National Research Council appointed a committee, the object of that committee, as is the object of all committees, to stimulate work on that particular subject, especially on the neglected phases, and secondarily to bring together—I do not mean secondarily—secondarily, to bring together the people working on different branches, and therefore I have been glad to speak to you, because I feel you are making one of the contributions of an understanding of relation, and I understand we are to make more progress if we can get together and appreciate the ways in which the others are working.

We are working more on the statistical side method, and what

you are giving us more of, all of us need, the experimental men are giving in the laboratory.

I want to set before you very briefly a few of the results that we are getting from statistical methods, and then I want to tell you what we plan, and how we want to ask your co-operation.

Recently, I had occasion to look over the statistics of Urban vs. Rural; you are all familiar with the facts. I had been familiar for a long time, but I had a surprise. I found that in New York City the urban death rate is decidely less than the rural rate. In Massachusetts, the same is true, in California, in Washington, and I believe there are one or two other cities. At first I supposed that was because those places show efficient departments of health.

A little further examination shows something else in there. I divided the states, for which statistics are available, into three groups. I took first a group where the cities are located on the sea coast, or on the lake shore, or in some cities where they have a special climatological advantage over the rural parts. New York is on the sea coast. People go to Atlantic City in the winter for their health. The same way with Buffalo. The breezes are an advantage in summer and in winter. The rural district in New York State lie in the center. The same in Massachusetts. I divided the three groups, those where cities had a marked advantage, those where they had a slight advantage, etc. Ohio is like that. Cleveland is on the lake, but Cincinnati is in the interior, and then the third group, the states where there was not particular advantage, like Missouri. St. Louis has no particular advantage over the rest of the state. They gave for the state, where the cities have an advantage, and advantage of about three or four per cent in the death rate, in favor of the cities; for the medium group about six or eight per cent advantage for the rural district; for the places where the city and country have approximately the same conditions, an advantage to the country of 25 per cent. There you have two factors. You have the city versus the country, and the city 25 per cent worse than the country, largely atmospheric, because our atmosphere is polluted with germs.

Our people live where they do not get enough sun and air, and it is far more the air than any other factor. We have the great factor of nearness to the sea coast, and diminution in extremes, and

the increased frequency of small changes, giving the city such an advantage, the sea coast city, that not only overcome the 25 per cent disadvantage which they have, but they get a three or four per cent advantage. That is one example of the kind of thing we need to study very fully.

Let me give you a second illustration. I brought some slides with me, and I made some maps, as to the distribution of civilization, in the opinion of fifty eminent authorities. I did this before the great war. Recently I have made some other maps, one of them being a map of health. I took the official records of all countries of Europe, reduced to a standard death rate, so the age and population has no effect, and furthermore I omitted children under one year, because the records are very bad and very variable from country to country, and I omitted old people, because the records are unreliable, and I made a map of that.

I made a third map. That map is based on records kept in factories of piece-work, and on the basis of work actually done by the piece-workers, under different conditions of outside temperature and humidity, and I made a map showing what would be the distribution of energy. Those three maps are so alike that if you did not know which was which and could not tell them apart.

Around the North Sea there is an area of high civilization, good health, high climatical energy. From the North Sea it diminishes. There is a little province of good conditions in Italy, the Black Sea and the Baltic. These are in the three maps. That is an extraordinary thing. Those maps are so much alike that you cannot question the relationship. You may say that I did not make the maps right, but supposing the maps are made right, they must be tested more fully. I think they are mathematically correct.

How can that relationship work? Just one way. Of course health is influenced by civilization, the higher civilized the people the more becomes their education on health. If people are strong and well they will advance more in civilization, but neither can touch climate; nothing you can do to civilization makes the climate different.

There is just one order in which that can be done, climate, health and civilization—I have made three of the United States, and

they are about as like as Europe. They show a high area in the northeastern country, high on the Pacific coast, low in the southeastern, and low in the Rocky Mountain states. In this health resort you have outdor air. These pictures I took were life insurance statistics, and there was less effect of outdoor air than you get when you have a health resort. That illustrates what we have. This investigation has not been completed, but so far as I have gone it shows some striking results.

We have had a good many investigations of influenza and all of us would like to know what caused it. It was a bacteria infection, and we agree something or other increased the virulence of the bacteria, and that is as far as we have gone. There is another phase to it. Granted that every city in the United States was bound to have that epidemic, have we any way of knowing, could we have known beforehand how severe that epidemic was going to be? I am beginning to think we could.

Dr. Pearl of Johns Hopkins made an investigation and worked out the corelations between the speed with which the epidemic growed to its maximum severity and other factors, the deaths, etc. He finds that they are very closely corelated with the proneness of the population to respiratory diseases and cardiac diseases, but he also finds a corelation with latitude. I have taken it and gone further. Instead of taking latitude I have looked into the conditions of the climate for a period of about three months before the epidemic came, but, as I say, this is not completed; as far as I can see there is going to come out a very strong corelation between the speed with which the epidemic reached its maximum severity and the degree of severity on one side, and the degree of stimulous that there was in the climatical conditions during the three months. That degree of stimulous is made by taking nine million deaths—there is a very strong corelation seemingly indicating that people sometimes acquire a certain degree of resistance to disease, and that resistance is the thing determining how severe our diseases shall be.

The National Research Council appointed a committee, and we want to ask you to co-operate and ask you to appoint a representative who shall be confirmed and represent the American Medical Society. It would not be a large committee. It is to be composed

of men who are actively at work, and who will work at these problems.

We have three things under way now, intensive investigation of deaths in New York City for a period of eight years, dividing them partly according to disease and age, and that is being helped by the New York Department of Health, the New York Life Insurance Company and the Metropolitan Insurance Company, and we hope soon to get from that some extremely helpful results.

A second thing we have is a study of people in health, and so we take factory workers as a means. We are getting records, very accurate records of atmospheric conditions in factories, and the amount of work done, and we are also planning one series of laboratory experiments, but we have not got far in that, because we have got to raise funds for it, and we are doing our best to get everybody together, so that we can work on these problems as one great problem.

We hope by these various methods which I have mentioned, and by all the work everybody has done, within a few years to agree upon a certain set of atmospheric conditions as the nearest approach we can get to the idea, and trying a hospital demonstration on a large scale. When I investigated the condition of humidity in the operating room, I found that when the humidity in the operating rooms averaged between four and five, the operations were much more successful than when under identical conditions otherwise when the atmosphere was dry and moist.

I want officially to ask this Association's co-operation, and I will not specify the method. I hope that you will consider it an official request, and we need these things discussed and talked about.

PALPATION IN THE OUTLINING OF ORGANS AND DETERMINING PATHOLOGICAL CONDITIONS CAUSING DIFFERENT DEGREES OF DENSITY IN THE SAME ORGAN: LIGHT, TOUCH, PALPATION.

By F. M. POTTENGER, M.D.

LOS ANGELES, CALIFORNIA.

Since physical examination of the chest was first practised, inspection and palpation have been considered to be of comparatively little importance. Recently, however, through a study of the motor and trophic reflexes from the lung and through the method described in this paper, inspection and palpation have assumed positions of great importance. In fact, when carefully and intelligently carried out, they will give a surprisingly accurate picture of underlying intrathoracic pathology.

My early study of the muscle changes in tuberculosis caused me to palpate chests with greater care than that which is usually employed. As a result of this I found that one could not only determine the changes in the soft tissues covering the thorax by palpation, but that he could determine the state of underlying viscera as well. I communicated this observation first to this society ([1]), and later described it more fully and more accurately in other papers, ([2], [3], [4], [5]).

My first observation was that I could outline the borders of the heart by a very light touch. Later I found that this could be extended to other organs and even to differentiating different kinds of pathology in such viscera as the lungs and pleura. The outlining of the heart by palpation I proved in connection with the orthodiagraph under Schwartz, of Vienna, and then extended the method to other intrathoracic and intraabdominal conditions.

In order to emphasize the lightness of palpation necessary to determine these differences in density, I designated the method as "Light Touch Palpation." Much information can be derived from a "touch so light that it scarcely indents the skin"; but I use various degrees of pressure in my every day clinical work, depending upon

the information desired. Many times a comprehensive idea of the pathology in a given thorax may be obtained by passing the tips of the fingers or the palmar surface of the fingers and hand over the surface of the chest. I usually stroke the chest from above downward in making this examination; but for more carefully differentiating pathology represented by different degrees of density in the lungs and pleura, I have personally found that I can do it best by proceding from areas of less density to those of greater density.

Some clinicians have failed to grasp the point that this is palpation and not percussion. Warnecke ([6]) in a recent discussion of Ebstein's "Tastperkussion" expresses a very common misunderstanding of this method as follows: "It is now necessary to examine into Pottenger's Light Touch Palpation. I do not recognize that any new principal is discovered in Pottenger's method, since it can only be 'Palpatory Percussion' in the sense in which Ebstein has described it. As already mentioned, one must expect to recognize different degrees of intensity to the touch under different conditions; so at times one will necessarily employ a much lighter touch than at other times, but I consider it entirely unnecessary to bring this forth under a new description. It is my opinion that every observer will agree that Pottenger's results can only be thought of as being obtained through 'Palpatory Percussion,' combined with sound percussion, and that they cannot be obtained through the simple laying on of hands upon the thorax."

Warnecke's assumption that the findings elicitated on feeling over the thorax are due to percussion, and that the information obtained is a result of the resistance to the finger combined with the tone emitted is not true. There is no sound emitted, neither is there any contact between the fingers and the thoracic wall more forceful than that which results from the usual laying on of hands. In fact, when palpating, I usually start either at the apex or base and often palpate a large portion of a lung without removing my fingers from the surface of the chest.

I am conversant with Ebstein's Palpatory Percussion ([7]), but Light Touch Palpation as described by me is totally different. There is no stroke at all. It consists of feeling and interpreting the varying degrees of resistance which are conveyed to the fingers.

If one will palpate a piece of board one-quarter of an inch thick and compare it with another piece two inches thick, the difference can readily be perceived. With practice one can learn to detect slighter and slighter differences in thickness. So it is in palpating the various organs of the body. At first only such gross changes as those between a solid organ as the liver or the heart, and an air containing organ such as the lung, may be detected, but with practice, as one becomes proficient, he may outline any organ or any pathological condition which alters the density of an organ by utilizing the sense of touch, the same as he can by the more complicated method of percussion.

Those who are not accustomed to make such examinations by palpation, but who are trained in percussion, will probably not agree with me that palpation is less complicated than percussion; but this disagreement is due to the fact that the things we know usually seem simple while those we do not know often seem to be complex and difficult to understand.

Palpation as herein described is carried out in such a manner as to give one the maximum amount of information from the use of the sense of touch. He simply feels. The surprise when one fully learns this art is that he feels so much and so deeply. It is so contrary to our usual grasp of the idea of feeling, which confines it to those superficial objects which come in direct contact with the nerves which give us our touch sense, that we are apt *a priori* to consider the determination of different degrees of density in the deeper structures as impossible. One of the chief ways, however, in which progress is made, is by doing something which has hitherto been considered as impossible.

Outlining deep organs and differentiating pathological conditions which cause differences in density in the same organ when the palpating finger does not come in contact with the organ involved seems impossible, but it is not; it is a regular procedure carried out every day by my assistant and myself in our clinical work. Hearts are outlined; shafting of the mediastinum is recognized; borders of the lungs are determined, infiltration, cavities and compensatory emphysema are diagnosed; hilus thickening, and the enlarged bronchial trunks are determined; and conditions in the pleura such

as fluid and air and pathological thickenings are recognized as accurately by feeling through the chest wall as can be done by percussion.

The chief advantage that palpation possesses over percussion is that it can be more accurately limited to the area examined. A percussion stroke over the thorax produces evidence of two kinds (1) through the sense of touch and (2) through the sense of hearing. The resistance and the tone emitted are both resultants of many factors, such as the amount and condition of the soft tissue; the size, shape and elasticity of the bony thorax; and the size, shape and actual condition which results from past and present pathological changes in the pleura, heart, mediastinal structures and lungs. Further these are altered by such things as pathological conditions in the abdomen; gas in the intestinal tract; the position, whether lying or sitting; the objects on which the patient lies or sits; and objects which are nearby. If each organ and each physical condition found in an organ had its particular tone and its particular resistance which was always characteristic of it, and these were never unduly modified by other conditions, then percussion would be only a matter of practice; but it is an individual matter differing with every patient. Few men are expert, yes comparatively few are above the average in percussing and interpreting the data obtained. Unfortunately because of having studied percussion most examiners are willing to offer an opinion based on this procedure, and they usually interpret these findings as though the pathology which they are seeking is responsible for all data obtained.

Percussion data, whether obtained by resistance or by sound, are records of normal conditions plus the past and the present pathological history of each individual, in the part examined, and must be so interpreted.

No matter how light the stroke, the sound emitted will be largely made up of elements which arise from structures outside of the organ or area percussed. The resistance is influenced in the same manner but is more definitely confined to the area under examination when the stroke is light. Teachers of percussion, recognizing the confusion which arises interpreting percussion sounds, and the extraneous factors which modify resistance when the stroke is heavy, have been gradually urging very light percussion. Turban ([8]) was

an early teacher of the value of the sense of resistance. I would suggest that accuracy often can be further increased by omitting the stroke entirely and simply feeling.

No one must think, however, that this method is without its difficulties and its errors. Nor do I suggest it to displace percussion, but rather to combine with percussion and thus increase the evidence in the case.

In instances of rigid thorax, in emphysematous chests, in thickened pleura with disease in the underlying lung, one's skill in examining will be taked in palpating the same as in percussion.

One of the sources of failure on the part of men who try to test out this method ,is that they expect to master it at once and without much difficulty. All examining procedures used in diagnosing chest conditions are difficult to learn and are mastered only after much practice. Nor must anyone think that he can learn this with less difficulty than he learned percussion; and, since it is not taught by teachers of physical diagnosis, but must be learned without a teacher by men who are more or less familiar with other methods, it is put to test at great disadvantage. Its value, however, is beyond question and I commend it to the patient consideration of those who wish to help add another valuable diagnostic measure to our too limited methods of finding out what pathology lies within the thorax. It can be used in examining all organs of the body, but it is in the chest that we are most in need of more accurate methods.

For those who are desirous of learning this method I would suggest three visceral borders as being easiest to make out: 1, the left border of the liver; 2, the upper border of the liver in the right axilla, and 3, the left border of the heart in the fourth interspace. In determining these borders one proceeds in palpating in each instance from a tissue of slight density to one of greater density, or the reverse. It is much easier to detect the border of a solid organ than to detect different degrees of density in the same tissue.

Palpation must often be done through the ribs, a thing which might at first seem impossible but which is not. One of the marvelous facts connected with palpation is that organs and various pathologic conditions in the same organ can be delimited through bone as well as thruogh soft tissue. The lung can be examined and

infiltration and cavities made out by palpating through the scapula. The caution should be given, however, when carefully examining for changes in density of tissues or for the border of an organ such as the liver or the heart to restrict palpation either to the intercostal spaces, or to the ribs, not to palpate from one to the other. If one passes from the intercostal spaces to the ribs or the reverse, it is difficult to accurately measure the difference in resistance which is due to differences in these superficial tissues, hence difficult to accurately judge of the density of the underlying tissue.

In order to determine the left border of the liver, the examiner should palpate over the lower ribs just below the heart, beginning well out toward the axilla and palpating slowly toward the median line. The normal left border of the liver lies under the left costal arch below and slightly to the left of the apex of the heart, and will be recognized as an increased resistance to the finger.

The upper border of the liver may be found by starting the palpation in the intercostal spaces high up in the right axilla and gradually approaching the position of the upper liver margin. I usually use two fingers either the first and second, or the second and third, and palpate two intercostal spaces at one time. Acute pleurisy either dry or with effusion and thickened pleura are the most common conditions which interfere with this procedure. The margin of the liver is recognized by an increased resistance to the fingers.

The left border of the heart in the fourth interspace is comparatively easy to find in normal chests, because one finds a fairly large volume of air containing lung tissue to compare with the dense muscle of the heart. It is very confusing in many chests, however, particularly those which have been the seat of influenza, pneumonia, or repeated attacks of bronchitis, which have left the patients with dense hilus tissues and thickened bronchial walls. These structures lie immediately to the left of the heart border and give an impression of increased density which can be readily mistaken for the heart. The same confusion exists in determining the right border. I personally, often have been confused by these conditions. They can be differentiated by care, but often prove to be very confusing to a beginner. An emphysematous left lung or a dense pulmonary infiltration is also confusing.

I would suggest that one learning this palpatory method should first outline the organs by percussion or follow on x-ray plate. This would make the beginner feel surer of his results.

After one has learned to outline and delimit such denser organs as the heart and liver, he will then begin to see the many possibilities which palpation offers. It is in determining the various pathologic states which attend pulmonary tuberculosis that I have found its greatest use.

Having learned to appreciate the resistance which is offered by normal lung tissue, one is then ready to note abnormalities. Healthy lung has a normal feeling as it is palpated through the thoracic wall the same as it has a more or less definite feeling and emits a more or less definite note on percussion and the same as it gives a more or less definite sound to the ear or stethoscope. While these data vary in different chests, yet we learn through experience that such and such data are within the range of normal variation.

The results obtained by palpating normal lungs will also vary; but one characteristic will always be the same—increased pressure over the deeper tissues will always fail to produce markedly increased resistance. My meaning can be ascertained by palpating over the heart or liver, or over the muscular portions of the leg and comparing with the lung. Increased pressure over the former meets markedly increased resistance; while over the latter, it elicits comparatively little change.

One must always bear in mind in palpation the same as in percussion, that the rigidity of the bony cage; the thickness and condition (normal, spasm or degeneration) of the muscles; the thickness or other condition of other soft parts; the thickness of the bony structure through which he is palpating; the nearness to the insertion of muscles; the nearness to the junction of the ribs with the sternum and vertebrae; and the relative amount of solid and air containing tissues, will alter the findings.

With these things in mind one is ready to examine the lungs by palpation. The examination must be systematically made. It is well to first palpate portions of the lung which are resonant. This often may be inferred from inspection, by noting those areas which move most freely, the motion appearing to be normal or above

normal in amount; or it may be determined by percussion and ausculation. Such areas are to furnish a comparative palpatory resistance by which other areas are to be judged. To one who can appreciate the difference between a solid and an air containing organ, the difference between an infiltrated and normal lung should be quite easy to differentiate.

Where evidence of pleural adhestions or other basal thickening is absent, I prefer to start at the base in the axillary region and examine each intercostal area systematically going from the axilla toward the median line, noting each change in resistance. In chests, either of tuberculous or non-tuberculous individuals which have been the seat of bronchial infections, one will usually note an increased resistance around the hilus extending out into the lung tissue and over the area lying to the left of the upper two-thirds of the heart and to the right of the sternum, and posteriorly several centimeters on each side of the vertebral column extending from the midcapular region toward and often continuing to the base. This will correspond to the dense shadows which are shown in the x-ray plate and are due to the thickenings which occur in the hilus tissues and bronchial walls.

In cases showing tuberculous infiltration, in palpating from healthy to diseased tissue (and this can usually be done by starting the examination in the lower intercostal spaces and examining each higher space in succession) one will notice an increased resistance when the lower border of the area of infiltration is reached. The resistance will vary with the difference in the pathological processes present. The extent and relative amount of active infiltration and scar tissue will cause differences which will be perceived by the palpating finger. Light infiltration shows very delicate changes in the resistance perceived, while dense infiltrations may at times feel much like such solid organs as the heart and liver or a large muscle mass. The fixing of the upper segments of the chest by spasm of the scaleni in early pulmonary inflammation is usually readily detected as an increased resistance.

Cavities usually form in the midst of a densely infiltrated area, and may be suspected because of this fact. On palpating over an area of infiltration which is marked by an increased resistance, cavity

is suspected by a sudden decrease in resistance. A decreased resistance surrounded by an area of increased resistance is probably one of the most dependable signs of cavity. Cavities are most easily palpated in the first and second interspaces anteriorly, although they may be detected in other areas even through the scapula if extra care is used. Error will sometimes occur in the second intercostal space in case it is wide; for under these circumstances the finger will sink deeply, because the resistance is less than normal.

Compensatory emphysema usually occurs in pulmonary tuberculosis as a result of prolonged coughing and when lung tissue has been destroyed and conditions are such that the intrathoracic space can be better filled by enlargement of pulmonary air cells than by such other compensatory measures as elevation of the diaphragm and contraction of the thoracic wall. It produces a relatively greater volume of air containing tissue, hence theoretically presents to the palpating fingers a feeling of resistance less than even that of the normal lung. Actually this is not true, for the enlargement of the air cells is usually accompanied by an increased tension and expansion of that portion of lung tissue involved which presses upon the inclosing thoracic cage causing increased tension of the intercostal tissues, and holding the thorax in a state of inspiration. This increased tension gives a feeling of increased resistance to palpation; but that it is not due to increased density may be readily determined by th fact that on heavier palpation—increased pressure—the resistance does not increase. A dense tissue when palpated lightly offers a sense of resistance which is increased by increased palpatory pressur; but an increased resistance over the lung which is due to increased tension fails to offer greater resistance on heavier pressure.

Pneumothorax either as a result of spontaneous rapture or artificial measures, if large, gives an increased tension with a decreased resistance on heavier pressure the same as compensatory emphysema. My meaning may be gained by palpating a balloon in different degrees of distention.

Pleural effusion, emphysema, and thickened pleura may be detected by palpation. They show as increased resistance to the palpating finger. The former two may cause wide and bulging inter-

costal spaces, while the latter is usually accompanied by a narrowing of the same.

Enlarged mediastinal glands and thickening about the hilus may be detected by palpation. In order to detect these I usually begin high up and posteriorly palpate with two fingers, one on either side of the spinal column. As one approaches the denser tissue the increased resistance may be noted.

The data obtained upon palpation as herein described are entirely aside from the changes which are noted in the muscles and subcutaneous tissues as a result of reflexes from the lung and pleura. Changes in soft tissues such as spasm of muscles, and degeneration of mucles and subcutaneous tissue, must always be taken into consideration, however, when palpating the deep tissues, because anything that changes the density and elasticity of the muscles and subcutaneous tissue influences to that extent all findings noted on palpating the deeper structures. I have purposely discussed this phase of palpation alone, in order to avoid the confusion that often comes to the mind of the reader when I discuss the outlining of organs and different pathological conditions in organs by touch at the same time that I discuss the changes which are perceived in the muscles and subcutaneous tissue as a result of reflex stimulation when the lung is inflamed. These two methods of examination are entirely different both as to the procedure in their execution and in the date obtained.

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DISCUSSION.

Dr. Babcock: I am intensely interested in what Dr. Pottenger said, and agree with him very much in some respects. Some people can do it, and some people can not, and furthermore there is unquestionably the great possible difference in the touch sense, or the sensitiveness of the fingers in one individual over another.

Now I have used palpation a good deal, and I think I detect some things that perhaps others do not, yet I do not claim to have a very sensitive touch.

The only illustration is what can be done by cultivation in certain people.

Dr. Pottenger said his left hand was more sensitive. In this girl's case her left middle finger, right middle finger, and the right forefinger, were the most sensitive. There is a heap in this sort of thing, and if we will cultivate it better, some people can acquire to a degree of nicety what others cannot.

A Member: That brings to my mind the early days when I was taught light touch over the abdomen, and I took very great interest in it. Later when I was teaching graduates, probably several hundred under my observation, and I could get no man to become interested in that, notwithstanding the fact I became enthusiastic. I could not get them to use it, in all of that group.

Dr. Elliott: I have used it for some time, and was taught to use it twenty-five years ago.

Dr. Mercur: At the last meeting of the American Congress of Physicians, four cases were brought in Minneapolis before Dr. Pottenger that he never saw. He put them up and palpated, and gave, without having seen them, a diagnosis of what he found. Immediately after that an x-ray picture was produced and read, and he was 100 per cent perfect. I would not have believed it if I had not seen it. In those cases he made 100 per cent diagnosis by inspection and palpation alone.

The President: Is there any further discussion?

Dr. Pottenger: The point Dr. Babcock made is true, but that holds in every other procedure. There is a great variation of men's efficiency in every diagnosis. It does not make any difference. My assistants all palpate the same

way, but some do it a great deal more accurately than others. But it is something that anybody can learn, but the point I make is this. If we study it, a certain percent of us will become efficient, and it is the same in any other thing, but I am sure I do not want this to displace anything. My purpose is to show by examination of this kind we can add something; we can improve our present methods and do better than ever before.

I am glad to hear what Dr. Ellott said. I did not know anybody described it before, and I am the first one I knew of, but I do not care who described it first; I have been working at it, and I know it is of great value.

In my method of palpation I begin over the lung and come toward the border (indicating). If I am going to outline the lung I always try to go to normal tissue, because normal tissue has a certain touch, the same as a certain note on percussion, and a certain degree of resistance. It will vary. I would begin here (indicating) and go up this way (indicating), and I do not take my hands off. I run my fingers like this (indicating). You will note the difference.

If I am going to outline a cavity, I feel deep, and go over it very carefully. Do not work too fast. Your brain cells will not work as fast as your fingers. I am careful, and make each feel, the right perception, and that is the method I use (indicating). I palpate very deep, and sometimes very, very light.

Dr. Litchfield: Do you get the absolute location?

Dr. Pottenger: The outer border of the heart.

Dr. Litchfield: The relative?

Dr. Pottenger: Yes.

Dr. Litchfield: And on the liver in the same way?

Dr. Pottenger: Yes. You will notice a difference when you come to the edge of the heart.

A STUDY OF THE BILE OBTAINED BY NON-SURGICAL BILIARY DRAINAGE, WITH ESPECIAL REFERENCE TO THE BACTERIOLOGY.

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It is not our intention in presenting this paper to attempt a discussion of the composition, chemistry and exertion of the bile. It is our purpose rather to try to point out the information of practical clinical importance that may be obtained from a study of the bile collected by means of duodenal drainage. Until recently such procedure would have been out of the question, for up to 1919, practically all studies upon the composition and bacteriology of the bile were made entirely upon bile obtained from the gall bladder at operations or at autopsy, or through post-operative biliary fistulae.

In 1919, B. B. Vincent Lyon (1) published a paper in which he showed how fresh bile may be obtained through duodenal drainage, basing this work of practical value upon a suggestion made by Meltzer (2) two years before in an article entitled "Disturbances of the Law of Contrary Innervation, as a Pathogenetic Factor in the Diseases of the Bile Ducts and Gall Bladder."

It will be recalled that in this communication Meltzer suggested the possibility of causing by reason of "crossed innervation" the paradoxical situation of relaxation of the sphimcter of the common duct and at the same time stimulation of the gall bladder and bile ducts to contract by the local application to the duodenum, by means of the duodenal tube, of a 25 per cent solution of magnesium sulphate. In making this suggestion Meltzer but elaborated upon the well proved physiological observation made over twenty years before by Dayon and Oddi. To Lyon, however, belongs the credit of first making practical use of these physiological and pharmacological facts. He showed that when through a duodenal tube hyperisotonic solutions of magnesium sulphate are brought in contact with the duodenal mucous membrane and are withdrawn before absorption

takes place, fresh bile may be obtained from the gall bladder and bile ducts, and further that this bile may be separated into three definite fraction, i.e., A, bile which comes from the common duct; B, bile which is of a different character and is derived from the gall bladder, and C, bile which is obtained from the hepatic ducts and bile capillaries and canniculae.

Abundant confirmation of his earlier work has been obtained by Lyon with an enormous series of cases, and has been further confirmed by a large number of other observers. Thus there has been established a comparatively simple technique by which with reasonable certainty, fresh bile from various portions of the bilary tract may be collected through the duodenal tube. It is not too much to say that the method has become firmly established, and must be looked upon as a well recognized and necessary additional method of gastri-intestional examination.

In passing it is perhaps fair to point out that the hypothesis advanced by Meltzer, and its further application by Lyon, has not been accorded uniform approval. There are those who question the validity of applying the law of crossed innervation, to the gall bladder and the muscle of Oddi. There are others who question whether the sphincter action of the muscle of Oddi is definitely established. Again specific action of magnesium sulphate in bringing about this relaxation and stimulation through intraduodenal excitation. Much stress is laid upon the fact that Meltzer's hypothesis is purely theoretical and has never been proven. The careful experimental work of Doyon and Oddi in 1894-1895 seems to refute this latter contention. A thorough discussion of these mooted points would here be out of place, but it is our belief based upon personal observation and experience with the method, in conjunction with careful clinical and experimental evidence, that the principle enunciated by Meltzer and put to practical use by Lyon, is in the main entirely reliable. Admirable clinical discussion of the entire question has been furnished by Smithies and Karshner, and Oleson ([3]).

During the past year transduodenal lavage and stimulation of the flow of bile by the introduction of hyperisotonic magnesium sulphate solution, has become almost a routine practice in our Gastro-intestinal Clinic at the Graduate School of the University of

Pennsylvania. In the course of this work we have met with the difficulties and disappointments from time to time, that have been common to all workers in this field. On the whole, however, our results have been encouraging and rarely has serious difficulty been experienced in introducing a duodenal tube into the duodenum. As a rule not over an hour and a half has been required to get the tube in place. We have not had the difficulties claimed by some, in determining the position of the duodenal tube. Without resorting to fluoroscopic examination we believe that the presence of the tube in the duodenum can be readily determined by the presence of the duodenal tug, i.e., when the tube is in the duodenum the plunger of the syringe attached to the free end of the tube will return to its position when traction is made upon it. Such is not the case when the tip is still in the stomach. Second, the appearance of pure yellow bile on aspiration. Third, failure to obtain water in the syringe after half a glass of water has been taken by mouth and fourth, when the tube is in the duodenum, 30 c. c. of warm water and allowed to run in and are then syphoned off by gravity, the water will return definitely bile stained.

On the whole, we have experienced but little difficulty in recognizing the free bile fraction referred to by Lyon, namely, the A, B, and C bile. Doubt has been entertained by some as to whether the dark bile alleged to come from the gall bladder is actually derived from this source. In this connection observations which are being carried out by one of us in conjunction with Dr. Eimann of the Bacteriological Laboratories of the Presbyterian Hospital of Philadelphia, and which await later publication, are suggestive. Estimation of the cholestrin content of the various specimens of bile conclusively shows that in the dark gall bladder bile, the amount of cholesterin is distinctly greater than that obtained from either the common duct of hepatic duct specimens. This increased cholesterin content results upon the concentration and stagnation of the bile, which takes place in the gall bladder. The remarkable ability of the gall bladder to concentrate the bile, and the restriction of this concentrating ability to the gall bladder, has been admirably shown by the work of Rous and McMaster (1). These authors have shown fluid is not withdrawn from the bile in the bile ducts whereas the

gall bladder is able to reduce the bulk of bile delivered to it as much as 10.8 times in 24 hours.

The technique employed by us for obtaining samples of bile for ordinary study, is essentially the same as that advocated by Lyon and now widely employed. The method is so well known that detailed discussion seems unnecessary.

The accumulated observations of many workers during the last couple of years leave no doubt but that valuable data are obtainable from bile collected by the transduodenal route, among which with a fair degree of accuracy, may be determined the state of the biliary tract. In order to be of value it is generally conceded that the study of the bile must be carried out when the specimens are fresh. These studied may be grouped under three headings. The gross appearance of the bile; miscroscopic appearance and its bacteriology.

Except when such examinations are carried out, the following conclusions are justified as to the state of the biliary passages.

1. If the bile, the A. bile obtained from the common bile duct, is darker than normal and more vicious, and shows the presence of a considerable number of epithelial cells, mucus leucocytes and bacteria, it is suggestive of chronic bile duct infection. When such infection is associated with stones, cholesterin crystals and the bile are increased, the bile frequently feels gritty.

2. When the B. bile derived from the gall bladder shows increased viscosity, is darker than normal and shows increased epithelial cells together with a large number of bacteria and leucocytes, and increased cholesterin crystals, an acute cholicystitis is probably present. When there is marked suppuration of the gall bladder, the number of leucocytes present may be so great as to be looked upon as pus. In violent acute inflammation a large number of red blood cells are frequently found. When in addition to an acute cholicystitis, gall stones exist, small calculi, gritty substances in the bile and a marked increase in cholesterin crystals is usually present. In chroniccholicystitis, the bile derived from the gall bladder is usually clear, shows flakes of suspended mucus, and a moderate increase in leucocytes. Frequently the presence of a number of bacteria may be recognized.

3. Failure to obtain the gall bladder bile after repeated stimu-

lation when the clear yellow bile derived from the bile ducts flows freely, strongly suggests the presence of cystic duct obstruction.

When after repeated stimulation no bile is obtainable, complete occlusion of the common bile duct may be inferred.

Disease of the biliary tract, therefore, depends upon careful observation of the viscosity, color of the bile, presence and absence of cellular elements such as epithelial cells, leucocytes, red blood cells, the amount of cholesterin present, the freedom of the bile from gritty particles, and finally the number and the varieties of bacteria present.

From a diagnostic, prognostic and therapeutic standpoint, obviously great interest centers about the bacteriological examination of the bile. A satisfactory technique for carrying out such examinations is obviously difficult, and many factors must be overcome before the result obtained can be regarded as reliable. In numerous instances we have attempted bacteriological studies of the bile. In an effort to have these observations as reliable as possible the following technique, after the method advocated by Lyon, has been adopted.

The night before the examination is to be made, the patient must brush the teeth thoroughly, the mouth and throat and nose are carefully washed by Lavoris followed by Liquor Antisepticus. From then on no further food or fluid is ingested until the tube is passed on the following morning on a fasting stomach. Prior to the passage of the tube, the teeth are again scrubbed, the nose, throat and mouth are thoroughly disinfected by Lavoried followed by Liquor Antisepticus. The duodenal tube having been rendered sterile by careful surgical sterilization, is then passed by an operator wearing sterile gloves and a sterile gown. As soon as the duodenal tube has reached the stomach, the stomach is carefully washed with distilled water until the return is entirely clear. It is next washed with a solution as Lavoris c. c. being diluted with 200 c. c. of sterile water. This lavage is followed by another lavage of sterile water. Then 250 c. c. of a 1 to 1000 Silvol solution is introduced into the stomach and removed. It is followed by another lavage with sterile water which is continued until the return flow is entirely clear. Such fluid or contents as is then remaining in the stomach is then cultured and the duodenal

tube allowed to pass into the duodenum. Before stimulation with magnesium sulphate a culture is made from the duodenal contents. Then stimulation of the duodenum is carried out as usual, with 75 c. c. of a 25 per cent of sterile magnesium sulphate solution. The bile is then obtained in the usual way, being segregated in the three fractions A, B and C bile from all of which separate cultures are made.

By following this method it is generally possible to render the upper alimentary tract as sterile as possible, and by taking cultures from the stomach and duodenum and possibly the mouth and throat before stimulation is made and the bile is aspirated, one is enabled to know which if any organisms are present in these structures before the flow of bile begins. Exceptionally the only organism obtained from the bile, is the same as those obtained from the stomach or duodenum before the bile has entered it. The organism found cannot be regarded as in any way peculiar to the bile. The cultures obtained from bile contain only the commoner mouth saprophytes such as the steptococcus salivarious, micrococcus catarrhalis, or the bacillus subtlis. No significance can be attached to the findings which must be looked upon as due to contamination. The organisms which ,however, are of importance and which have been repeatedly isolated by numerous observers, include streptococci, especially hemolyticus, the staphylococcus aureas and albas, the bacillus typhosis and the colon bacillus. From the somewhat meagre published results of bacteriological examination of the bile thus obtained, it is surprising to note the frequency with which some observers hemolytic streptococci. It is also interesting to note that pneomococci have been reported by some. It seems somewhat incomprehensible in view of the well known bile soluble property of pnumococci.

It must be admitted that the technical difficulties, as well as the rather meagre knowledge at present available as to the bacteriology of the stomach, duodenum and biliary passages often makes the bacteriological study of the bile distinctly unsatisfactory.

Elaborate and careful experimental studies on the bacterial statis and germicidial properties of the bile, have been carried out by Neilson and Meyer ([5]). Their experiments were largely carried out

with test tubes and included a variety of biles from different animals, such as cats, goats, rats, dogs, oxen, sheep, pigs and a few observations on bile obtained from man. The human specimens were obtained wholly from cholicystectomized bladders by aspiration from laparotomy. Their observations were made largely upon organisms of the typhoid dysentery paratyphoid group, and the ziblio of chlorea. In the specimens of bile obtained from man they found that the typhoid bacilli grew well and remained visible for more than ten days, whereas in the ziblio cholera, it required eight days before the sterilization was complete. They concluded that the rigor of bacterial growth in average human cystic bile, is noteworthy. They further discovered that proliferation is somewhat influenced by artificially inducing hydronion concentration, but that complete suspension of growth only occurs at a PH which in all probability never occurs in a human body. The importance of their paper, so far as man is concerned, has largely to do with the influence of cystic bile on the human typhoid carrier state.

It was originally our intention to discuss the bacteriological findings in a series of cases that exhibited disease of the biliary tract or gall bladder, but in the earlier portion of our work so much difficulty was experienced with the technique, that the results obtained were regarded as too unreliable for permanent record. We have had an opportunity of carrying out some successful and interesting bacteriological observations on a limited number of cases, and in order to demonstrate the importance of the bacteriological findings in the bile, these cases will be briefly reported.

Case I.

Mr. C. P., Jr. Male, aged 60. The first of January this case suffered from gastro intestinal disturbances, associated with marked jaundice. A diagnosis was made by his physician of acute catarrhal jaundice. In the course of two weeks the jaundice entirely cleared up.

The patient first came under observation three weeks after the onset of his jaundice. At that time there was no evidence of jaundice on physical examination. Stools contained bile, and the urine was free of bile. He exhibited, however, considerable pallor. There was some elevation of blood pressure, he suffered from a distinct asthenia,

coated tongue, and loss of appetite. Examination of the urine showed the presence of considerable quantities of albumin, numerous hyaline and granular casts, and red blood cells. He was admitted to the hospital apparently suffering from acute exacerbation of a chronic.

Haemateria became more marked, and the anemia progressed. Careful search was made for a focus of infection that might be held responsible for the acute renal manifestations. All posible foci of infection were eliminated except the gall bladder, over which there was a slight persistent tenderness. With the history of a recent attack of jaundice ,it was decided that the gall bladder was in all probability the focus of infection responsible for his progressive anemi and his physical symptoms. Three weeks after coming under observation his haemoglobin was 55%; his red cells 2,860,000 and his leucocytes 17,100. At this juncture duodenal drainage was instituted. A considerable quantity of dark green gall bladder bile was obtained. In this bile was found numerous flakes of mucus and sediment, epithelial cells, in three of the specimens of gall bladder bile, pus was present. On culture on two separate occasions, staphylococcus aureus and albus were found in pure culture and the A, B, and C bile, but these organisms were not present in the cultures taken from the stomach or from the duodenum before stimulation was resorted to. It was also interesting to note that several specimens of urine collected at about the same time and examined at a different laboratory, also gave pure cultures of the same organism. There seemed, therefore, to be some justification for the assumption that the bladder and renal infection were the result of the same organism. Owing to a steadily falling blood count, the patient was given a transfusion of 500 c. c. of blood. Some slight improvement followed this procedure, although the blood count did not noticeably increase. The most marked subjective improvement was a biliary drainage, which was practiced once a week. A vaccine was prepared from the strains of streptococcus aureus and albus isolated from the bile. During the past month the patient has been receiving the vaccine every four days. The haematuria has cleared up, the renal symptoms have subsided, the blood count has shown marked improvement and subjectively the patient shows every indication of recovery.

Case II.

Mr. E. D., aged 52, in whom a diagnosis of duodenal ulcer and cholicystitis was made. The condition of the gall bladder was discovered in the course of doing a drainage, during a course of two weeks treatment with duodenal feeding. In this patient the microscopic study of the bile showed a considerable number of pus cells, bile stained epithelial cells, many short motile bacilli, a few pigment crystals and quantities of amorphous bile salts. Bile from the gall bladder was obtained by the aseptic technique of Lyon cultures showed a luxurious growth of colon bacilli. An autogenous vaccine of colon bacilli was prepared, and injections were given at intervals of four days up 1,000,000,000 bacilli. At the same time weekly drainage was practiced. The original culture was obtained on January 13th. Following the above outline treatment, on March 23rd a second culture was obtained by the same technique. At this time the patient was symptomatically greatly improved. Gastro intestinal symptoms had disappeared.

Case III.

Mrs. G. B., aged 35. A patient in the service of Dr. Pemberton suffering from chronic arthritis. After exhaustive search for the foci of infection in the course of which the tonsils were removed, and some teeth extracted without improvement, non-surgical drainage of the biliary tract was instituted. The B bile was a deep black. Was vicid, contained many shreds and particles and on culture showed many streoptococcus hemolyticus and staphylococcus albus in luxurious growths. In view of these findings, the gall bladder was regarded as the probable focus of infection and was removed. At operation no stones were found but a definite chronic cholicystitis existed. Cultures taken from the gall bladder at operation showed hemolytic streptococcus and staphlyococcus albus. Confirmation by surgical means of the findings obtained by the transduodenal drainage are interesting and significant of the value of the latter method. Bi-weekly biliary drainage was instituted in this case. Two months after the original culture was obtained from the bile, bile cultures were again made following the Lyon antiseptic technique. This time the staphylococcus albus alone was present. Streptococcus had dis-

appeared. An autogenous vaccine was prepared and given. A month later the biliary drainage was discontinued and the patient at that time was showing marked improvement, her temperature was practically normal and she was able to be up and walk about.

Case IV.

Miss K., aged 60. This patient was suffering from a chronic arthritis. Her tonsils had been removed without result. Biliary drainage was instituted, cultures were obtained according to the Lyon technique. A non-hemolytic steptococcus was isolated from the B bile. A vaccine was made and is being administered. The patient is still under observation and although improving it is too early to know the ultimate result.

Case V.

Mr. M., aged 53. The patient gave a history of recurring attacks of slight jaundice and so-called biliousness. These latter he had ever since his youth. Biliary drainage was instituted and cultures made from the bile. In this instance although the bile showed staphylococcus albus in the bile, non-hemolytic streprotocci and unidentified micrococci were also found in the stomach cultures. The results therefore obtained from the biliary culture were too uncertain to justify any therapeutic procedure based upon these findings without further checking the results. This case is cited to emphasize the importance of culturing the stomach, even though it is believed to be sterile, since even though the same organism has been discovered in the bile, they could not be regarded as significant since they had also been isolated from the gastric culture.

These cases are cited in order to point out the possibility of accurate bacteriologic study of the bile by means of biliary drainage, and also to again emphasize the tree frequently forgotten rôles that the gall bladder plays in producing focal infection.

The checking by operative findings of the results of biliary drainage, in Case III, is a gratifying evidence of the reliability of bacteriological studies made by this technique. A much more striking example, however, of the value of pre-operative duodenal findings in comparison with the lesion found at operation, is furnished by Whipple ([6]) who in a series of 27 cases on whom careful studies

of the biliary tract were made by means of duodenal drainage before operation, found a striking coincidence in the operative findings with those obtained by a study of the bile before operation.

In carrying out bacteriological studies of the bile, a caution which has been rightly emphasized by Lyon, should be pointed out. Lyon insists upon the importance of replanting from the original cross culture if one is used, in six hours. If this is not done, a rapidly growing colon bacillus may render it extremely difficult to isolate the slower growing steptococcus, should such an organism be present. In order to render the bacteriologic examination doubly safe, bile is planted on gauze cultures during the biliary drainage, and is transplanted within twelve hours. In addition to this all the bile withdrawn is collected in twelve bottles separated into the A, B, and C biles, and streaks are made from these biles on solid media at once. By following this method the isolation of these organisms will be facilitated.

Conclusions.

1. We believe that the method of biliary drainage instituted by Lyon, and based upon the hypothesis of Meltzer is a useful and practical procedure. That there is every reason for the belief that the biles obtained in this way are derived from the common bile duct, the gall bladder, the hepatic duct and biliary capillaries.

2. In our experience the diseased condition of the gall bladder and bile ducts can be recognized in this way by a chemical and bacteriological study of the bile, which in a pathological condition show alterations which are significant.

3. The bacteriological study of the bile derived in this way, is feasible and is of importance, but in order to be successful, a rather elaborate, time-consuming technique is essential.

4. If such a technique is carried out, the bacteriological findings are reliable and are a guide to therapeutic procedure.

Finally, however, it should be emphasized that it is our feeling that the information obtained by transduodenal lavage is only a diagnostic adjunct, and in no sense should take the place of careful history taking and complete physical examination. It is only when the findings derived from biliary drainage are taken in conjunction with a complete clinical study of the case that accurate conclusions

can be drawn. Our knowledge of the bacteriology of the biliary and upper gastro intestinal tract, is still so meagre that a great deal of work and much careful observation will be required before this phase of the subject is placed upon an entirely satisfactory basis.

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DISCUSSION.

The President: The paper is now open for discussion.

Dr. Daland: Mr. President and gentlemen, I congratulate Dr. Piersol in having summarized in such a satisfactory way the status of gall bladder and biliary drainage, from the various standpoints to which he refers. I believe there are quite a number of examples illustrating what he said.

Referring to the difficulties in regard to the bile, in regard to its effects upon microorganisms, the effect upon pneumococcus, and from what he states his conclusion is most encouraging, opening up an entirely new avenue of information, diagnosis, and is an additional adjunct, as he states, to our existing knowledge.

I have nothing to offer. I am interested in the subject, and I would like to ask Dr. Piersol one question, and that is, in these cases of gall bladder disease, where he recovered a specific organism, and the gall bladder was the focus of infection, as to whether or not there existed elsewhere a focus that preceded the infection of the gall bladder?

The President: Is there further discussion on this paper?

A Member: I have gained a good deal from the discussion of Dr. Piersol, and I think his is a very able paper.

A year ago before this Association I reported some thirty cases in which biliary drainage was done, which was followed by operation. In these thirty cases there were twenty-six cases in which bile was absent, and in all of those cases we found either an obstruction to the cystic duct, and the gall bladder was full of stones, and did not contain any of this bile. That was helpful in quite a number of ways, as we not only got a better idea of diagnosis, but we were more easily able to handle a patient and convince him that an operation was necessary.

I am very glad to say that in the past year our experience has been the same, practically the same, that is, that the absence of B bile convinces us that the cystic duct, the gall bladder itself—great care must be exercised—this must

be done very carefully, and the less we check ourselves up by giving large doses of belladonna and repeating the test, we could very easily be misled, because sometimes we do not get the B bile at the first sitting, and we have gone over these cases two and three times, also seeing from the x-ray whether our tube is in the proper place, and in checking up in that way we felt that we have accomplished something. I am very glad to have heard this paper, and I have the assurance that it is worth while going on with the work.

THE PRESIDENT: Is there further discussion of Dr. Piersol's paper?

Dr. Piersol, will you close the discussion?

DR. PIERSOL: In answer to Dr. Daland's question, so far as we were able to determine, we were not able to discover any foci of infection other than the gall bladder.

DIAGNOSIS OF FOCAL INFECTION.

By JUDSON DALAND, M.D.,

PILADELPHIA, PA.

The diagnosis of focal infection took on an increase, much because we more fully realized the relations with the things that produce it, and also the cause of its relationship with other processes, such as tuberculosis, and certain conditions which require operative procedure.

In order to spare time, I shall make no reference at all to the diagnostic signs of focal infection, but shall confine myself to diagnosis and the state of that focus: in other words, I shall confine myself to the diagnosis of the focus of infection, and its location and probable condition in which that focus is. The focus condition in chronic, acute or latent form, we are familiar with, and they need no discussion. There are a certain number of acute focal infections that are primarily chronic, and the acute is looked upon as being acute, and one of the most conspicuous examples of acute focus of infection would be in the case of a tooth that has been prepared for filling and has been promptly filled, and speedily thereafter arises a painful swelling, and sometimes fever.

In these cases there is very little doubt in my mind that there was a pre-existence of infection around about the devitalized tooth. The sub-acute processes are exceedingly common, and likewise, of course, the chronic are the most common. We recognize how they become a focus, and I want to call attention to the fact that very, very frequently the existence of sinisitis, more especially when it involves the sphenoid, is overlooked. I do not mean it is overlooked only by the ordinary observer. It is frequently overlooked by the expert.

There is a reason for a negative report in the presence of sinisitis; it is not due to ignorance or lack of skill on the part of the expert, but very frequently due to the intrinsic difficulty of making a definite diagnosis in those cases where a few cells are involved.

We have, however, in recent years become so advancd, so far as the x-ray is concerned, that now slight variations in shadows can be made out, and these become an important factor in sinisitis. Given an increase in shadow over a suspected area, this calls the attention of the expert to this region, and when it is further examined, he immediately discovers that this is the result of a previously existing disease, which is passed on to an evidence of the existence of an active focus of infection, which may produce this invasion.

There are some men throughout the country who say that they are prepared to give you a definite opinion of sinisitis. There is not a single man in the world capable of excluding sinisitis by one examination. A number of examinations are necessary before you get it —the evidence from which you are sure you have sinisitis. I am dwelling on this negative side, because in my earlier work I was impressed because I felt perfectly certain, and a good many men joined with me, not infrequently I was able to diagnose the focus.

If you are unable to diagnose the focus you are not doing much good for your patient. In adults I have seen cases of focal infection in connection with the mastoid, and I know it occurs in children and at different ages, but I say that in my own particular experience I have not had the good fortune to meet with the number of cases that do exist.

We must keep in our minds very clearly the possibility of the existence of the mastoid as a sort of focal infection, even in the absence of the ordinary symptoms and signs, because this, like the others, is apt to exist. That is the important point, that you can have an active focus of infection in this and the other locality, and the particular local symptoms that are manifested are not clear, and therefore the repeated examinations in these regions are necessary.

I want to say a few words on the subject of the tonsil. I am doing it purely from the standpoint of the internist.

I was in the habit of attaching much importance to the history of a preceding tonsillectomy, and I still maintain that when you are searching to make a diagnosis of a location of focus of infection, such a history has considerable importance, more especially when it is associated with enlarged glands, which are often hard—that shows that preceding it as some time in the past there has been an infec-

tion which involved secondarily the lymphatics, and having occurred in the past you can look upon that tonsil as a support.

Then, again, in a great many of these cases, where you have an idea that there has been a chronic, long-standing infection of the tonsil, you do not find lympathic gland enlargements, because they are so located, or the individual is so constituted, that you can not discover them by palpation. Sufficient care is not exercised in attempting to find them sometimes. The tonsil when you look at it does not give you usually very much information except that you can see a hyperproby, and see it is small, or see that it seems normal in size, or you may see some milky fluid or cheesy material, and it may give you neither, and when you are through with the examination you might think there is no doubt about the existence of tonsilitis or a diseased tonsil, but the real question you are concerned with has a bearing upon the systematic manifestations that you are contending with. We must realize that when we look at a tonsil we only see one aspect of it, and you must look very carefully, and although your eyes may not be as good as a specialist, nevertheless his eyes cannot penetrate any further than yours, and therefore an absolute and definite negative statement regarding the infectivity by an expert may not disturb your equilibrium.

The other point about the tonsil is the attachment of anterior and posterior—I desire to say with emphasis, upon a matter of importance in the diagnosis of infectivity of a chronically diseased tonsil, that the expulsion from the crips of any cheesey material—that is always a matter of suspicion, but no proof, and the laryngologist has introduced a new word into our nomenclature which I think is interesting, "criptic," and I think the remark is "cryptic."

The examination of these crips, after they have been emptied out by a loop of platinum going to the bottom, not infrequently reveals a streptococcus, and this streptococcus may be of the viridans, or it may be hemolyptic, or the ordinary variety, and in judgment constitutes an additional point when you can get such an organism out of such a tonsil.

Our President intimated that I have a few seconds only, and I, in those few minutes cannot do what I had a desire to do, to touch upon that subject, the most difficult of subjects, the diagnosis of the

location of focus of infection, and I may state that the general rule is safe, foci infection—and not only do we have multiple foci of infection, but very frequently an assciation of infection, and furthermore it is not infrequent to have also a sionist infection, and we may have three reasons. The number may be quite enormous.

I have frequently diagnosed one, and not the other two. I have diagnosed two, and sometimes not the third. We should remove all foci. He may be temporarily well after the removal of one focus.

I will say a few words, whereas I desire to say much if I had the time, regarding the question of determination on your part, whether a devitalized tooth needs extraction. I beg of you to recall in your study of teeth, from all the different angles, that negative evidence today is just as weak as it ever was. All evidence that may be described as weak, but negative evidence may be termed as very weak. It is positivve evidence that we desire, and in going over devitalized teeth, in the absence of an x-ray evidence of a molar for instance, we will say, is to be looked upon as one part of the cumulative evidence you are to meet, in order to determine what you are going to do. The x-ray is physical, and we can blot out and get a negative x-ray plate. There are conditions in the crotch of a tooth where there is a focus of infection where the x-ray is made, and do not exclude dental infection. I would like to say in going over this question that it is necessary to do the following complex act. The proper study of this question requires the combined or team work on the part of the physician and the bacteriologist. Here again we get into trouble. Bacteriologists do their work in a routine way. For this work it is absolutely necessary that the work be done at the time of the extraction, or at the time of the removal of the tonsil, to obtain material for cultural studies, the bringing together of these men to work simultaneously on these cases is vital.

I regret the time is so short that I cannot go further.

DISCUSSION.

Dr. Rochester: I was very much interested in his presentation, and feel I cannot say too much on the possibility of infections of the cells, particularly the ethmoid. I have had three cases of a disease of the lung which we could not—two of them were acute asthmatic, and one chronic bronchitis, and sent these cases for examination to one of our excellent men, and he sent word that there was nothing the trouble with the upper air passages, and I sent them to

one of our radiographers, and he sent back the same report, and I got into the same conversation with another radiographer, and I told him there must be trouble; I felt sure there was. One of the reasons I felt there was, was this: These patients, all of them, had at certain times during the day, not at all times, a definite secretion on the posterior wall of their pharynx, and their noses were perfectly clear, and the evidence was that there was no involvment of other signs.

This man made several studies and finally found the trouble in the ethmoid cells. These cases had trouble with the ethmoid cells, and after that was corrected they got well, whereas before all the treatment did no good.

There is another source of infection which Dr. Daland did not indicate, and that is the appendix.

I have had several cases of sub-acute nephritis, which did not clear up at all until the appendix was removed, and then it got well.

Dr. Babcock: I have already said too much in this meting, but I would like to dwell on this particular point.

In cases of chronic infected tonsils, it is very frequent that you do not get a history of tonsilitis. You may ask your people, "Have you ever had tonsilitis?" and he will say, "No." "Have you had a sore throat?"; "Oh, yes."

It is well known that acute tonsilitis does not often produce pain in children, and therefore tonsilitis is overlooked in children because of the absence of pain.

I would like to say also that the combination of chronicly infected tonsils, and chronicaly infected teeth is very frequent the combination of the two.

I could talk for a long time on focal infections within the abdomen. I want to emphasize particularly, as has the last speaker, the necessity of examining the abdomen carefully with reference to the appendix, but also to the gall bladder. I know I have had cases which have been entirely relieved by the recognition of a chronic appendicitis, and the removal of the appendix, and I wish further to say that I have come to be suspicious of the gall bladder or appendix in all patients who complain every now and then of stomach trouble, and I often investigate the two structures, the appendix and gall bladder with particular care.

Dr. Forster: I want to say that Dr. Babcock spoke of infection of the abdomen.

Last year in reporting three cases, prostate and similar cases, Dr. Sewell said that focal infection very frequently stopped at the neck. I hoped that Dr. Daland would have the time to discuss these other focal infections. Then again, particularly in the appendix, there is room for allusion to the connection between focal infection in various parts of the body, certainly focal infection in the tonsil, in the teeth, or in the sinisitis, may very often account for a focal infection in the gastro intestinal tract.

There is one other point I would like to ask Dr. Daland about, in connection with the teeth. In looking up a case I came across the statement that teeth are always infected. That seems to be going a little further than some of those

which are particularly interested in focal infection of the teeth, and I wonder what Dr. Daland's experience is.

DR. DALAND: Very frequently they are not infected.

I desire to call attention to the fact that not infrequently a remnant remains, something remains, and I do not know why it should, but this remnant, although it may be very small, culture sometimes shows the pure hemolytic streptococcus, and I want to reiterate what has been said in this Association in a former communication, that we must never for one minute allow our minds to become concerned with the size of the focus, which is of no importance. A part of a tonsil no longer than a pea is able to give you all kinds of secondary manifestation. A good many will forget that. They seem to be mentally unable to grasp the idea.

In the second place I would like to report that after another year's experience, I have been enormously aided in diagnosing the latent cases of focal infection, and in a great many other cases, by the study of the leucocytres—that picture of lymphocytic focus has been of enormous assistance to me in determining whether this or that thing or this thing should be done, whereas if it is local we will wait.

CLINICAL ANALYSIS OF FORTY CASES OF BACTERIEMIA.

By RAYMOND CLARK, M.D.,

Brooklyn, New York.

(*From the Medical Service of the Brooklyn Hospital, Brooklyn, N. Y.*)

The special object in presenting a clinical study on bacteriemia to this Society is to place before you, for discussion, clinical data obtained from the analysis of forty consecutive cases of bacteriemia which occurred in the general service of the Brooklyn Hospital during the last five years. The cases presented include only those where the laboratory has recorded a positive culture of the blood for micro-organisms.

A general hospital like ours is not equipped to turn out records for detailed scientific deductive purposes as are the hospitals especially organized and equipped for research work. I trust therefore you will bear with me if some of my deductions are based upon clinical premises. However, I want to state that clinical reports from our general hospitals throughout the conutry play an important part in furthering the knowledge obtained from our highly scientific research institutions. The scope of this paper is limited to the commoner micro-organisms and no attempt has been made to make a complete study of the bacteriology of blood stream invasion. The definition of bacteriemia simply implies the presence of micro-organisms in the circulating blood. However, in the cases presented, in addition to the bacteriemia, all the patients showed, to a greater or lesser degree, a systemic response on the part of the body tissues to the infecting organism, manifested by the clinical picture in each patient.

Dr. Louis Nerb, our bacteriologist, who has been with us for the past five years and who has shown an unusual interest in co-ordinating the laboratory and ward work of the hospital, has kindly outlined the technique for identifying the micro-organisms found in this series.

Blood Culture Technique.

To obtain the best results one must familiarize oneself with the nature of the infection suspected in order to culture in suitable media.

The blood is carefully taken under usual asceptic precautions, preferably from the anticubital space of arm. The needle is plunged into the vein and about 10 c.c. of blood withdrawn. The tip of the needle is flamed quickly, 5 c.c. of blood divided into the individual culture media and the rest is put into a large sterile test tube containing 3 c.c. of a two per cent sodium citrate in normal saline. The latter procedure was found very successful, inasmuch as the blood is prevented from clotting by the action of the sodium citrate and can be plated out later in the laboratory to a suitable medium. The action of the sodium citrate does not inhibit the growth of the organism. This was tried out by reculturing the same blood after standing for two weeks in the ice chest when practically the same number of colonies were obtained on culture as in the fresh specimen.

Streptococcus Pyogenes:

Have a slightly alkaline glucose broth (beef infusion) and three glucose agar tubes at 40 C. Into each of these plant 2 c.c. of whole blood. The agar with whole blood is plated immediately in Petri dishes and allowed to cool. Two of the Petri dishes are set aside for the partial oxygen tension method, by which excellent results are obtained. The method is as follows:

Take covers of Petri dishes of identically the same size as the others where whole blood with glucose agar was planted. Into each of these pour a thin layer of ordinary agar and allow to solidify. A small loopful of B. Subtilis is transplanted in the center of the media. These covers are placed over inverted Petri dishes containing the blood agar. At contact of covers, fasten with narrow strip of adhesive tape and seal with paraffin. All the cultures are put into the incubator at 37.5 C.

In bouillon the pyogenic streptococcus grow rapidly in long tortuous chains and have a tendency to form flakes which sink to the bottom. Any further growth will usually cease on account of the

rapid formation of lactic acid; this may be obviated by the addition of one percent sterile CaCO. On glucose agar cultures the colonies show a definite characteristic. The colonies of streptococcus longus or hemolyticus will appear with a light halo of hemolysis. Streptococcus viridans has a definite green color. Very frequently, even though there is no growth in broth, and glucose agar, a luxuriant growth of colonies is obtained with the partial oxygen tension method. For further identification of the organism (on account of the morphological similarity to pneumococcus) the colonies are transplanted to inulin medium. Streptococcus will not ferment and solidify inulin—pneumococcus will.

Bacillus Typhosus:

Two flasks of bouillon of 100 c.c. each are inoculated. The first with 1 c.c. and the other with 2 c.c. of blood. Then 2.5 c.c. of blood are put into a sterile bile, peptone and glycerine medium and incubated. After 12 hours, 24 hours, 48 hours, and after three days transplants are made from the broth and bile media on Conradi-Drigalski or Endo media plates. The typhoid colonies will appear red on Conradi-Drigalski and Endo media. Some of these are scraped off emulsified in broth and observed for their morphology and motility. Lastly, an agglutination test is done with known typhoid immune serum.

Bacillus Coli:

The procedure is practically the same as in typhoid. The suspected colonies which appear on Conradi-Drigalski, as yellow colonies are carefully picked out and inoculated into carbohydrate serum water media (dextrose, lactose, and saccharose) in fermentation tubes. Colon Communis Virus will produce gas and acid in dextose and lactose, whereas in saccharose there is no gas formation and the litmus remains unchanged. Colon Communior will produce gas and acid in all three media.

Staphylococcus Albus and Aureus:

The culturing is done in the same manner as for the previously mentioned organism. Staphylococcus will produce distinct cloud-

iness in broth and small round colonies in blood agar without hemolysis. The organisms are easily identified with the microscope by their morphology. For further identification, broth and colonies from the blood agar are transplanted in serum agar. Staphylococcus aureus will appear with a bright yellow color, staphylococcus albus colonies remain white.

Diplococcus Pneumoniae:

In 100 c.c. bouillon flasks are inoculated respectively 1 and 2 c.c. of blood. To two glucose agar tubes are added 1 c.c. of blood each and plated in Petri dishes. The growth will appear in nutrient broth usually within twenty-four hours and leads to slight clouding of the fluid, which will gradually disappear and sink to the bottom. In the Petri dishes the white colonies appear with a slight halo of hemolysis. The colonies are fished out and transplanted to Hiss serum water inulin for differentiation of streptococci. The inulin serum water will be acidified and the coagulated by the growth of pneumococcus. Streptococci will not ferment inulin. To differentiate the types of pneumococcus, a slight emulsion of the colonies is made in sterile saline solution, inoculated into a mouse and a regular pneumococcus typing is done.

Bacillus Aerogenous Capsulatus (B. Welchii):

The blood is taken in the same manner as previously mentioned.

1. Glucose agar tubes are liquified by heating and cooled to 40 C, then 1 c.c. of blood is inoculated, well mixed, the agar is allowed to solidify. The cotton plug is burned and pushed about 2 c.m. into the opening of the tube. Then a layer of about 1 c.m. of pyrogallic acid crystals is firmly parked on top of the cotton, and 1 c.c. of strong NaOH poured over the crystals and the tube closed with a well fitted rubber cork.

2. A large tube of glucose broth is put into boiling water for fifteen minutes, then rapidly cooled to exclude the oxygen, 1 c.c. of blood inoculated, slightly agitated and 2 c.c. of sterile albuline oil floated on top of the broth, to prevent oxygen from entering into the media.

B. Welchii grow very rapidly, and usually a luxuriant growth is

obtained from 18 to 24 hours. The glucose agar will show many characteristic gas bubbles. The broth shows a distinct cloudiness and a row of gas bubbles surrounds the free surface of the medium. Smears are taken from the broth and a capsule stain made. For definite identification a few c.c. of the broth cultures are injected into the ear vein of a rabbit. The animal is killed after five minutes and placed in the incubator for 12 to 18 hours. The rabbit will be found enormously distended with gas. The abdomen of the animal is tapped with a trocar, and the gas collected in a test tube by downward displacement. On ignition, it will explode with a typical hydrogen bark. The autopsy of the rabbit shows most of the organs are riddled with gas bubbles and especially the liver, which has the appearance of a rubber sponge and a distinct odor of butyric acid.

Smears are made from the liver and from the heart's blood. The bacilli will be seen with a beautiful capsule.

Preparation of Magnesium Sulphate Solution:

Numerous experiments with various strength solution of magnesium sulphate have showed us that .1% solution will kill streptococcus in vitro within a half hour. Stronger and weaker solutions were found to be too strong or too weak. In the preparation of this .1% solution the patient's blood is the dilutent. Hence, the patient's weight is found, 1/15th of which weight is blood, e.g. patient weighs 150 lbs. (68.1 kilos), the patient's blood will be 10 lbs. (4.5 kilos, or 4500 gms.) Hence, that patient must get 4.5 gms. of magnesium sulphate made up in a 2 or 2½% solution of sterile water in order to make the whole blood a .1% solution of magnesium sulphate.

Comment.

The Value of the Partial Tension Method:

Our experience has shown us that this method is three times as efficient as the ordinary aerobic technique, i.e. this method will usually give three to four times as heavy a growth as the ordinary method, and also, the partial oxygen tension method will frequently show a growth where the ordinary one remains sterile. Incidentally, W. W. Oliver and C. C. Perkins, who originated this method and have done so much work in perfecting this method, found, to quote

from their paper Partial Tension Streptococci and Vaccine Preparation, "The adaptation of this strain (of streptococcus) to growth at partial tension is suggested by the fact that its isolation was effected only by incubation of the cultures under diminished oxygen tension, as well as by the fact that subsequent partial tension transplants yielded a moderately heavy growth, whereas, aerobic transplants yielded, at best, only a few, tiny scattered colonies and, in some cases, no growth at all."

We have found this diminished oxygen tension method to be of further great value in the making up of autogenous vaccines. When the organism is grown by this method a luxuriant growth is obtained within 24-48 hours. This enables the patient to receive the vaccine within two days whereas the old method of growing the organism would require about seven days in the preparation of such a vaccine.

So far as we know this method of figuring out this strength of magnesium sulphate solution has not been tried elsewhere.

TABLE OF CLINICAL STATISTICS.

Diagnosis of Infections	No. of Cases	Died	Recovered	Improved	Unimproved	Recovery %
STREPTOCOCCUS VIRIDANS						
Endocarditis, acute	3	3	..	..	..	0
Endocarditis, subacute	5	3	..	..	2	0
Cellulitis of Thorax	1	1	..	..	..	0
Abortion, subacute Salpingitis	1	..	1	..	..	100
STREPTOCOCCUS HEMOLYTICUS						
Rheumatism, acute articular	1	..	1	..	..	100
Abortion, strepto. septicemia	2	1	1	..	..	50
STREPTOCOCCUS BREVIS						
Endocarditis, acute	2	2	..	..	..	0
Streptococcic Septicemia	1	1	..	..	..	0
Rheumatism, acute articular	1	..	1	..	..	100
STAPHYLOCOCCUS AUREUS						
Gumma of Sternum, infected	1	..	1	..	..	100
Arthritis, acute suppurative	1	1	..	..	..	0
Arthritis, subacute	1	..	..	1	..	0
Pelvic Cellulitis, acute	1	1	..	..	..	0
Lung Abscess	1	..	..	1	..	0
STAPHYLOCOCCUS ALBUS						
Staphylococcic Septicemia	1	1	..	..	..	0
BACILLUS COLI COMMUNIS						
Cholecystitis, acute gangrenous	2	1	..	1	..	0

Pneumococcus Type II, III, IV						
Pneumonia, acute lobar	2	2	..	..	..	0
Pneumonia, broncho	6	2	4	..	..	66
Pneumococcic Septicemia, Gen.	1	1	..	..	..	0
Bacillus Typhose						
Typhoid Fever	5	..	4	1	..	80
Bacillus Aerogenes Capsulatus (Welch's gas bacillus)						
Cellulitis of Left Arm	1	..	1	..	..	100

TABLE OF GENERAL SUMMARY.

Organisms	No. of Cases	Recovered	Improved	Unimproved	Died	Mortality %
Streptococcus viridans	10	1	..	2	7	70
Streptococcus hemolyticus	3	1	1	..	1	33
Streptococcus brevis	4	1	..	..	3	75
Pneumococcus type II	1	..	..	..	1	100
Pneumococcus type III	2	..	..	..	2	100
Pneumococcus type IV	6	4	..	..	2	33
Bacillus Coli communis	2	..	1	..	1	50
Staphylococcus aureus	5	1	2	..	2	40
Staphylococcus albus	1	..	..	..	1	100
Bacillus Typhosus	5	4	1	..	..	0
Bacillus Aerogenes capsulatus	1	1	..	..	..	0

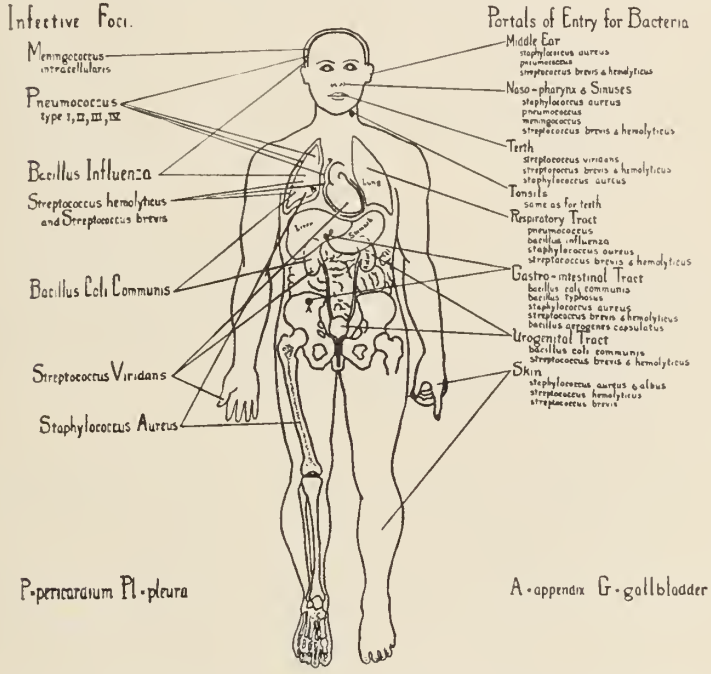
Clinical Statistics:

For the study of these conditions two tabulations have been evolved. First the table of clinical statistics which shows each type of infecting micro-organism, with the different clinical entities grouped under each organism, giving also the number of cases and the end result of each clinical entity in per cent recovered. The second, a summary table showing each infecting organism with its number of cases and the patient's end result expressed in mortality percentage. These tables will be utilized in the further analysis.

Portals of Entry: Infective Foci and Complicating Lesions,

Of all the portals of entry for bacteria into the body, the teeth, with their adjacent structures, and the tonsils have attracted by far, the greatest amount of discussion. Rosenow, and many dental surgeons, believe that the lesions about the teeth and surrounding structures which are encapsulated are the most potential and dangerous form of dental sepsis; far more serious than the dental sepsis associated with advanced dental caries where there is drainage through the pulp substance into the oral cavity, or dental caries with

surrounding lymphatic involvement which acts as a barrier between the focus of infection and the blood stream. It has been my experience that it is the more or less innocent appearing tooth, in which it is difficult to state positively from the x-ray shadow that there is an apical infection, and yet with an encapsulated focus of infection, well walled off and its contents under pressure, that is the real cause of our clinical manifestations. The tonsils likewise harbor microorganisms in a similar manner as do the teeth and it is my belief that, all other portals of entry for focal infection having been



excluded, it is far wiser to have a tonsillectomy done and remove an innocent tonsil now and then, than to allow the patient to suffer for months as the result of focal infection of the tonsil.

In order to rapidly visualize these conditions, I have devised a schematic diagram of the body representing the portals of entry for bacteria on the one side, and the infective or metastatic foci, as the result of a bacteriemia, on the other, and have tabulated under each

portal of entry the bacteria which frequently enter through that portal and, by directing lines, have called attention to the commoner infective foci of each micro-organism.

The Streptococcus Viridans Infections:

The portals of entry were as follows: Cases 1, 2, 24, were through the tonsils; cases 21 and 31 were through the teeth; case 33 was through the urogenital tract; cases 16, 18, 23, 40, were through undetermined portals. Seven of the cases were in females, three in males. The age of the patient ranged between eighteen and forty-six. Seven of the cases showed mitral valve lesion, two cases of mitral and aortic vegetation, and case 33 died before there was clinical evidence of endocardial involvement. The complicating lesions were: Septic metastetic foci in skin, kidney, spleen, mesentery and brain, cellulitis of the thoracic wall; broncho-pneumonia, glomerulo-nephritis and severe secondary anaemia. Case 33, where the portal of entry was through the urogenital tract with a subacute salpingitis, with no apparent endocardial involvement, is the only case in which I can report a recovery. It is evident that when the endocardium once becomes involved the patient's chance for recovery is nil; this mocro-organism shows a marked predilection for the mitral valve, a well recognized clinical observation.

The Streptococcus Hemolyticus Infections:

The portals of entry were as follows: Case 6 through the tonsil; cases 8 and 39 were through the urogenital tract. Two of the cases were in females, one in the male. The patient's age ranged between twenty-eight and forty-eight years. The complicating lesions were: Multiple non-suppurative arthritis, acute suppurative arthritis, acute suppurative peritonitis and secondary anaemia. Cases 8 and 39 were associated with sepsis following induced abortion. None of the patients showed endocardial lesions, and it is my experience that this organism seldom, if ever, is the cause of endocarditis. This micro-organism, on the other hand, shows a predilection for the peritoneum, pleura and pericardium. The end result in these three patients was unusually good, case 6 recovered, case 8 was discharged from the hospital as improved, (a follow up shows her to

be in excellent health on April 15, 1922), case 39 died. This is a much higher percentage of recovery than is usual.

The Streptococcus Brevis Infections:

The portals of entry were as follows: Cases 4 and 5 were through the respiratory tract; case 22 through the tonsils and case 7 the portal was undetermined. Three of the cases were females, one male. The age of the patients ranged between seventeen and forty-four. The complicating lesions were: Acute endocarditis with mitral valve lesion, multiple non-suppurative arthritis, cellulitis of the neck, and general septicemia. This organism infected the endocardium, mitral valve, in two of the cases. The end result shows that cases 7 and 22 died of general sepsis, case 4 was discharged as improved, but a follow up shows that she died in September 1920, and case 5 made a complete recovery.

The Staphylococcus Aureus Infections:

The portals of entry were as follows: Cases 3 and 25 were through the skin, case 27 through the respiratory tract, cases 20 and 32 the portal was undetermined. Four of the patients were males and one female. The age of the patients ranged between eight and fifty-seven years. The complicating lesions were: Broncho-pneumonia, acute suppurative arthritis, pelvic cellulitis, lung abscess, non-suppurative arthritis, sero-fibrinous pleurisy, and general septicemia. This organism is a normal inhabitant of the skin and mucous surfaces, hence these are common portals of entry. The tendency is to produce localizing metastatic suppurative foci as is demonstrated in this series, all of the cases requiring surgical interference. The end result of these patients was as follows: Cases 20 and 25 died from general sepsis, cases 27 and 32 markedly improved, while case 3 completely recovered.

The Staphylococcus Albus Infection:

Of this type of infection I report only one case, case 34. The portal of entry was through the tonsil. The patient was a female, age twenty-one years. The complicating lesions were: Toxic jaundice, multiple abscesses in right kidney, infective petechial foci

in visceral, pericardial and endocardial surfaces. These findings were obtained at autopsy and by microscopic examination of the tissue. This organism, likewise, is a normal inhabitant of the skin and mucous surfaces. On account of its rapid elimination, normally, from the blood stream it is seldom the etiological factor for general sepsis; however, when this organism is a septic factor the end result of the infection is usually fatal, and this case substantiates that view.

The Bacillus Coli Communis Infection:

The portals of entry were as follows: Case 36, through the orogenital tract; case 38, through the gastro-intestinal tract (gall-bladder). The patients were both males and their ages were 29 and 42 years. The complicating lesions were: Acute suppurative peritonitis, broncho-pneumonia, abscess left parotid (Staphylococcic), toxic jaundice. This organism is quite capable of producing a general sepsis as is well exemplified in case 36—with recovery from the sepsis but leaving the patient with a damaged left kidney, pyelonephrosis. Case 38 died at the end of the seventh day from his peritonitis, undoubtedly due to a mixed infection.

The Pneumococcus Infections:

The portal of entry in all the cases was through the respiratory tract, except case 37 which was a general pneumococcic septicemia and the portal of entry was undetermined. Seven of the patients were males and two were females. The age of the patients ranged between eighteen and forty-six years. The complicating lesions were: Acute fibrinous pleurisy, sero-fibrinous pleurisy, acute suppurative pleurisy, toxic myocarditis, pneumococci septicemia, pericarditis, and septic petechial foci in skin. Case 37 was the only patient that presented any clinical evidence of septic emboli in the skin. Cases 9, 10, 11, 12 ,and 37 were of types II, III, IV; all manifested a marked septic clinical picture and all died. Cases 13, 14, 15, 17, were all of type IV; they showed a mild degree of sepsis and all fully recovered. It is of interest to note that in case 13 the sputum showed type III, which would indicate that he was suffering from a type of pneumococcus usually fatal, but the blood culture showed type IV, the type which was probably responsible for his infection.

The Bacillus Typhosus Infections:

The portal of entry in all the cases was through the gastro-intestinal tract; this is purely a clinical observation. Four cases occurred in males and one in the female. The ages varied between sixteen and twenty-nine years. The complicating lesions were: Acute bronchitis, toxic myocarditis, congestion of liver, spleen and kidneys, and typhoid carrier. The bacillus typhosus was found in the blood stream on the 8th, 9th, 13th, 19th, and 22nd day of the disease; the first day of the disease dating from the patient's first symptoms. The cases were under observation between 41 and 64 days. Case 29, a typhoid carrier, was discharged as improved, all others recovered.

The Bacillus Aerogenes Capsulatus Infections:

Case 30, male, fifty years of age and a tailor by occupation. The portal of entry was through a needle prick in the skin of the finger. The bacteriemia was transitory, due probably to the massive doses of specific serum (Welch-Tetanus) which were promptly administered, intravenously, together with the early amputation of the left upper extremity; no remote complications; patient recovered.

Treatment.

This includes both medical and surgical proceedures. In all cases, whenever it was possible, the focus of infection was surgically treated, as by removal or incision and drainage.

1. General:

All patients received, more or less, the usual hospital routine as to diet, palliative measures, and drugs. None of these measures however had any specific action, as far as could be determined, on the course of the infection.

2. Vaccine Therapy:

Three patients with streptococcic viridans infection, and one patient with pneumococcic type II infection, received specific vaccine treatment; the end result of all these patients was fatal. One patient with streptococcic hemolytic infection, one with staphylococcic

aureus infection, and one with B. coli infection, received specific vaccine therapy; these patients were all discharged from the hospital as improved.

It seems as if vaccine therapy seldom has any value in infections of acute fulminating type, whereas in low grade, subacute or chronic forms they show some therapeutic value.

3. Serum Therapy:

One patient, case 22, with a diagnosis of acute endocarditis due to the streptococcus brevis, was given, intravenously, a total of 250 c.c. of antistreptococcic serum. The case terminated fatally.

One patient, case 30, with a diagnosis of cellulitis of arm due to bacillus aerogenes capsulatus (Welch's gas bacillus), received intravenously a total of 380 c.c. of specific Welch-tetanus serum. This patient made a complete recovery.

It is the experience of the writer however that the anti-streptococcic serum is of some value as a therapeutic agent in cases of severe streptococcal infection. The use of the specific Welch-tetanus serum was thoroughly tested out in our recent World War and found to be of real therapeutic value.

4. Salvarsan:

One patient, case 37, with a diagnosis of general pneumococcic septicemia due to type IV organism, received several intravenous injections of salvarsan at intervals without any apparent effect; the patient died.

5. Magnesium Sulphate Solution:

Three patients, cases 1, 16, and 31, with a diagnosis of endocarditis due to the streptococcus viridans, received one intravenous injection each of 200 c.c. to 250 c.c. of magnesium sulphate solution, at intervals of six and twelve days; all four of these patients died.

One patient, case 8, with a diagnosis of abortion with sepsis due to streptococcic hemolyticus and without clinical evidence of endocarditis, received one intravenous injection of 150 c.c. of magnesium sulphate solution; the patient was discharged from the hospital,

improved. A follow up notation of April 15, 1922, states her condition as recovered.

One patient, case 4, with a diagnosis of acute endocarditis due to the streptococcus brevis organism, received one intravenous injection of 250 c.c. of magnesium sulphate solution; the patient was discharged from the hospital, improved. A follow up notation shows that she died September 1920, as a result of her endocarditis.

One patient, case 5, with a diagnosis of acute articular rheumatism due to the streptococcus brevis organism without clinical signs of endocardial involvment, received two intravenous injections of 150 c.c. of magnesium sulphate solution; the patient was discharged from the hospital as recovered.

All of the patients receiving the magnesium sulphate solution showed varying degrees of clinical reaction as, a drop in the temperature, sweating, chilly feelings and in some cases a chill, cardiovascular depression, nausea and a feeling of general weakness; these conditions lasting from twenty-four to forty-eight hours. In no case were these reactions alarming.

It has been our experience that magnesium sulphate solution, when introduced into the blood stream of a strength sufficient to make a dilution of .1% after it has been mixed with the total volume of blood, is of therapeutic value only in certain cases of sepsis, and these cases are those in which we find septicemia associated with a bacteriemia before embolic infective foci, especially in the endocardium, have occurred. The explanation of why this agent does not give better results, after infective foci, especially in the endocardium, have occurred, is, possibly, because the magnesium salt is incapable of sterilizing the metastatic focus as it apparently sterilizes the blood stream.

General Comments.

It should be borne in mind that in a series of cases of this kind where the number is small, few reliable deductions can be made. The forty cases showed deaths of twenty, a mortality of 50 per cent. These figures correspond closely to those of others who have made similar clinical studies.

The infections due to the Streptococcus viridans and brevis,

with endocardial involvement, the Staphylococcus albus, and the Pneumococcus type II and III all show an excessively high mortality. I believe that the Streptococcus hemolyticus should be added to this group, but the mortality in this series was unusually low, 33 per cent, also that infection due to the Pneumococcus type II shows a much higher mortality than is usually found.

The intravenous administration of magnesium sulphate solution proved of therapeutic value only in patients suffering from sepsis before embolic infective foci have occurred in the endocardium.

Realizing that most of the infections, especially those in which the endocardium is involved, gain entrance into the blood stream through recognized portals, it hardly seems right to conclude this study without some mention of prophylaxis. Those of us who are engaged in general diagnosis must realize that our survey and the advice given to the patient are both incomplete, unless, whether or not the patient is suffering as the result of focal infection, all the portals of entry for bacteria have been considered as potential or actual sources of sepsis.

Streptococcus Viridans Infection.

Case No. 1.—Medical Service.

C. D., female, age 46, single, school teacher, U. S., admitted Feb. 16, 1917.

Past History: Never strong and healthy; frequent attacks of tonsillitis; at age of 26 years suffered from attack of peritonsillar abscess, following this ill with "rheumatism," of joints especially; severe typhoid fever in June 1916; no illness since until present trouble.

Present Illness: Began two months ago, when she was obliged to give up teaching on account of mental and bodily fatigue; both ankles were swollen, red, and tender at this time, this swelling lasted only two days. She now complains of night sweats for the past few weeks; appetite is poor; some cough with muco-purulent expectoration. Loss of twenty pounds in last six years. No gastro-intestinal symptoms.

Physical Examination: General survey shows pale woman, poorly nourished, propped up in bed; skin not pigmented, no

petechial spots; many carious teeth; tonsils not apparently enlarged; thyroid normal. Heart is hypertrophied to left to 14 cm., right border 4 cm. from midsternal line; presystolic thrill and presystolic murmurs at apex; systolic murmur at apex transmitted to left; aortic areas negative. Lungs normal save for few rales over right base posteriorly. Abdomen—No points of tenderness; liver enlarged and smooth, extends to level of umbilicus; spleen 4 cm. below costal margin. Extremities show no joint involvement, no oedema; knee-jerks active; fundus (eye) is normal. Urianalysis: Faint trace of albumin, otherwise normal. Leucocytes 13,500, polys. 72%, Hb. 45%. Blood pressure: Systolic 100, diastolic 60. Temperature 100, pulse 110, respiration 20 per minute. Renal phthalein test: 28 for two hours. Wassermann: Negative. Blood culture positive for Streptococcus viridans on April 12, 1917; was negative on three previous examinations.

Clinical Course in Hospital: She was in the hospital fifty-five days, during which time the temperature averaged around 100 in P.M. with tendency to subnormal in A.M., on several occasions it went up to 102 in the P. M.; the pulse fluctuated between 80 and 110; the respirations never going above normal. Petechial areas appeared on left hand for the first time February 28th; others appeared on her extremities on March 13th and March 26th. Patient gradually lost strength on account of poor appetite and her mental stability became disturbed; she left hospital, by request, unimproved. The blood count on March 14th was Hb. 55%, red cell count 4,500,000, white cell count 9,100, polys. 60%. The urine showed no sign of a renal lesion.

Treatment: High caloric soft diet: digitalis was used for general cardiac support; hypo. administration of iron and arsenic for the anaemia; aspirin for the pain as was needed; sodium bromide for restlessness; out of doors on the porch as weather would permit; March 26, 1917, 250 c.c. magnesium sulphate sol. given intravenously, the temperature, pulse and respiration made no change in their course after the injection, no constitutional reaction.

Diagnosis: Subacute endocarditis, secondary anaemia.

Remarks: Portal of entry probably tonsils and heart valve

became involved during subsequent rheumatic attacks. Discharged, unimproved.

Follow up notation: Patient died on September 19, 1917.

Case No. 2.—Medical Service.

J. R., female, age 30, married, U. S., admitted March 13, 1917.

Past History: Rheumatism at age of 12 years, no subsequent attack; subsequent frequent attacks of tonsillitis. Usually enjoys good health.

Present Illness: Well until three months ago when she suffered from short illness of influenza; since then chest pains, loss of muscular strength, general malaise, precardial pains, occasional night sweats. No dyspnoea; no respiratory or gastro-intestinal symptoms.

Physical Examination: General survey shows well nourished woman; skin clear; does not appear acutely ill; thyroid normal; teeth appear normal, none in upper jaw; reflexes, knee and eye, normal. Lungs are negative. Heart is hypertrophied to 12 cm. to left of midsternal line, normal to the right; loud systolic at apex and transmitted to left. Abdomen—No masses or tenderness; liver and spleen of normal size. Extremities negative, no petechial spots. Urinalysis: Negative. Leucocytes 12,500, polys. 79%. Wassermann: Negative. Temperature 99, pulse 90, respirations 30 per minute. Blood pressure: Systolic 100, diastolic 60. Blood culture positive for Streptococcus viridans, on March 25, 1917.

Clinical Course in Hospital: Patient remained in hospital 26 days, during which time the temperature fluctuated between 98 and 101, pulse varied between 60 and 100, respiration 18 to 30.

Treatment: Soft nutritious diet. Open air porch most of the time while in hospital.

Diagnosis: Subacute endocarditis.

Remarks: No subsequent history has been obtained. Discharged, unimproved.

Case No. 16.—Medical Service.

M. B., female, age 37, married, Ireland, admitted May 25, 1919.

Past History: Negative.

Present Illness: Patient's condition was semi-comatose on admission and any reliable history was never obtained.

Physical Examination: General survey revealed patient apathe-

tic, lying with eyes closed and only aroused with difficulty; skin bathed in profuse perspiration; pulse rapid and weak; lungs show area of patchy consolidation posteriorly, especially in left lower; heart shows soft blowing systolic at apex; abdomen reveals tenderness in right lower quadrant, liver and spleen not enlarged; extremities negative; no petechia. Urinalysis: Heavy trace of albumin, hyaline and granular casts, pus cells, red blood cells; temperature 100, pulse 120, respirations 25 per minute; leucocytes 8,200, polys. 74%, Hb. 70%. Cystoscopy on May 25, 1919: Bladder urine shows moderate number of pus cells, hyaline and granular casts; right kidney numerous red blood cells, many pus cells, few hyaline casts; left kidney, many red blood cells, moderate number pus cells, few hyaline casts. Culture of bladder urine, sterile. Blood culture positive for Streptococcus viridans, on May 27, 1919.

Clinical Course in Hospital: Patient was in hospital five days. The highest temperature was, just before death, 104.5, pulse 140, respirations 40 per minute.

Treatment: Diet of general fluids. On May 28th magnesium sulphate sol., 250 c.c. of a 3% sol., was given intravenously; the pulse slowed from 120 to 96 per minute soon after the injection, then within half an hour returned to a rate of 112; no other reaction. Hypodermoclysis of saline solution six hours after the injection of the magnesium sulphate sol.

Diagnosis: Endocarditis, acute. Complications — Bronchopneumonia; multiple septic infarcts in kidney.

Remarks: Portal of entry, unknown. Presented the clinical picture of profound sepsis. Died May 29, 1919. No autopsy.

Case No. 18.—Medical Service.

J. Z., male, age 23, married, U. S., admitted June 20, 1919.

Past History: "Rheumatism as a child followed by heart trouble" and chorea at age of ten years.

Present Illness: Came to hospital complaining of gastro-intestinal disturbances for past few months, especially "belching" of gas after meals, and dyspnoea; a feeling of general muscular weakness.

Physical Examination: General survey shows nutrition fair; pale; no skin eruption; head and neck negative, save for several carious teeth and hypertrophied tonsils; lungs show few scattered

musical rales, otherwise negative, heart hypertrophied to left; apex beat just outside linea mammallaris; presystolic thrill felt over the mitral area; presystolic and systolic murmur heard at the apex; pulmonic second accentuated; slight diastolic murmur over aortic area but not transmitted along the sternum; rhythm is regular; abdomen—distended, some dullness in the flanks; liver enlarger to 4 in. below free costal border; spleen not felt; no other masses or points of tenderness; extremities negative. Urinalysis: Negative. Blood pressure: Systolic 140, diastolic 60. Pheno-sulpho-thalein 40% for two hours. Wassermann: Negative. Blood culture positive for Streptococcus viridans on June 23, 1919.

Clinical Course in Hospital: He was in the hospital four days when he signed his release. Temperature was subnormal on admission but the next day it reached 99, pulse was rapid—120 to 140 per minute, respiration averaged 30; the total daily urinary output was low—20 to 30 oz. but no renal lesion could be made out.

Treatment: Salt free light diet; cardiac stimulation, especially tr. digitalis and saline cathartics.

Diagnosis: Subacute infectious endocarditis with complication of cardiac decompensation.

Remarks: Patient did not remain in hospital long enough to start any treatment, but went home as soon as the decompensation was improved. Social Service reports that patient died on August 4, 1919.

Case No. 21.—Medical Service.

C. S., female, age 18, single, stock-girl, U. S., admitted October 22, 1919.

Past History: Negative.

Present Illness: Four months ago began losing weight, has lost 12 pounds; one month ago began to have chilly feelings, night sweats, and, about 11 A.M., headaches; no respiratory or digestive symptoms.

Physical Examination: General survey shows patient emaciated; anaemic; neuro-muscular reflexes normal; no lymphatic enlargement; thyroid not palpable; skin clear; heart enlarged to linea mammallaris; blowing systolic at apex, transmitted to left and heard behind; lungs are negative; liver 3 in. below and spleen 2 in.

below free costal margin. X-ray of chest is negative. X-ray of teeth shows pus pockets at apices of three. Blood pressure: Systolic 120, diastolic 60. Hb. 55%, erythocytes 3,130,000 leucocytes 17,500, polys. 82%. Blood culture positive for Streptococcus viridans, on January 10, 1920.

Clinical Course in Hospital: Patient was in hospital 135 days, during which time the temperature ranged between 100-103, pulse 100-120, respirations 20-30. Three blood cultures, taken each month after admission, were sterile. Faint trace of albumin with granular casts appeared in the urine at times. The blood culture became positive after three months in the hospital, the lowest hemoglobin reading was 54%. The complications which developed were: Glomeruli-nephritis, recognized on 57th day; on December 30, 1919, signs of metastatic emboli in spleen; following this signs of mesenteric emboli.

Treatment: Carious teeth were extracted on December 16, 1919, with slight fall in temperature for a few days, no ill results. Unfortunately no cultures were made. Vaccine, stock combined VanCotts, was started on November 24, 1919, and continued for several weeks without any apparent improvement. January 11, 1920 ,intravenous transfusion of 200 c.c. of 2.5% sol. magnesium sulphate was given; following this the temperature rose to 104, pulse and respiration remained as before, and at the end of 24 hours the temperature continued much lower for one week, when it again ran as usual. On January 17, 1920, intravenous injection of 200 c.c. magnesium sulphate sol. was again given; the temperature rose to 103 for 24 hours when it dropped to 100 and continued down for four days, gradually returning to an average of 102; the pulse increased in rate from 110 to 120 per minute. On January 29, 1920, third introvenous magnesium sulphate sol. was given, this last had little or no effect on temperature. There were no marked reactions to the administration of the magnesium sulphate except profuse sweating with some depression. There was a fall in the temperature for several days following the magnesium injection.

Diagnosis: Endocarditis.

Remarks: Patient was discharged from the hospital as the family wished her home during the last of her illness. Portal of entry

through the teeth abscesses. Patient died two weeks after leaving hospital. No autopsy.

Case No. 23.—Medical Service.

C. B., male, age 45, married, guard, Ireland, admitted December 7, 1919.

Past History: Attack of "rheumatism" in 1906. Has known he has diabetes mellitus for last four years, during which time he has been on a diabetic diet. Has been able to work up to present time.

Present Illness: Began suddenly on December 3, 1919, with stabbing pain in left chest, chilly feelings, dry cough, some fever and severe headache; sputum at times has been rusty.

Physical Examination: General survey shows man acutely ill; anxious expression; dyspnoea marked; no evidence of cerebro-spinal disease; mass of enlarged glands in left axilla, tender and somewhat reddened, no fluctuation; marked tenderness with bogginess over whole left chest especially in axillary region; left lung shows signs of patchy consolidation especially behind; heart is hypertrophied to linea mammallaris, blowing systolic at apex; aortic second accentuated; Abdomen—Liver enlarged 4 cm. below costal border and not tender; spleen palpable 3 cm. below free costal margin. Urinalysis: 1025, no albumin, marked reduction to Fehling's sol., no casts. Leucocytes 9,400, polys. 57%. X-ray of chest is negative. Wassermann reaction two plus. Blood chemistry: CO combining power of blood 34.2; Urea in 100 c.c. of blood 28.03 mgs.; Creatinine in 100 c.c. of blood 1.7 mgs.; Sugar in 100 c.c. of blood .5%. Blood culture positive for Streptococcus viridans, on December 11, 1919.

Clinical Course in Hospital: Patient was in hospital five days; he was admitted with a temperature of 104, pulse 120, respiration 30 per minute, this rate continued for three days when it rose to temperature 105, pulse 160, respiration 50; he gradually showed signs of increasing sepsis with cardiac failure and died on the fifth day after admission.

Treatment: Incision and drainage to lesion in right chest wall followed by wet dressings to both chest and axilla; cardiac stimulants given, digitalis, camphor in oil; Murphy drip of 5% bicar-

bonate soda sol. and large doses of bicarbonate soda by mouth, this was especially directed to combat the acidosis.

Diagnosis: Cellulitis of left side of thorax. Septicemia (Streptococcus viridans). Diabetes mellitus.

Remarks: The patient did not show a severe acidosis as the result of the diabetes, but was overwhelmingly toxic as a result of his chest wall cellulitis and septic broncho-pneumonia. Patient died. No autopsy allowed.

Case No. 24.—Medical Service.

E. J., male, age 26, single, statistician, U. S., admitted May 21, 1920.

Past History: Pneumonia followed by empyema 20 years ago, good recovery. In 1906, following an attack of acute tonsillitis, was ill with acute rheumatic fever, leaving him with damaged mitral and aortic valves. In 1913 another attack, much worse than the previous, of rheumatic fever lasting six weeks; some cardiac decompensation occurred at this time. Still another attack, in 1915, of less severity although was quite protracted. Twelve years ago he had his tonsils removed, this was in 1910.

Present Illness: Began on March 12, 1922. He was taken ill while in England and the physician diagnosed his illness as influenza. He was given a vaccine derived from a throat culture, without improvement; just before leaving for America, the patient noticed tender red spots appearing at the tips of his fingers and toes, which would disappear in two or three days.

Physical Examination: General survey shows young man lying in bed acutely ill, but not suffering pain; head negative; teeth many filled, otherwise negative; tongue coated; no thyroid enlargement; vessels of neck show a marked, rapid pulsation, veins somewhat distended; lungs negative save for dysfunction of left chest, as result of empyema. Heart—diffuse pulsation over the entire precordium, apex in 6th interspace 11 c.m. from midsternal line, and right border 5.5 cm. to the right of same line; presystolic thrill is felt over the mitral area; there is a presystolic harsh murmur heard at the mitral area, which is ascending in quality to systole; it is followed by a softer systolic murmum which is transmitted out into the axilla; at the aortic area there is a systolic harsh murmur, transmitted into

vessels of neck; this is followed by a soft, blowing, diastolic murmur. Abdomen: There is a tenderness in the epigastric region and especially over a small area near the top of the left (9th) costal cartilage; liver normal; spleen 2 cm. below free costal margin. Extremities, some clubbing of fingers, no petechial spots. Pulse rapid, water-hammer type. Temperature 101, pulse 110, respiration 25. Urinalysis: Negative. Leucocytes 8,000, polys. 85%. Blood culture positive for Streptococcus viridans, on May 23, 1920.

Diagnosis: Endocarditis, subacute infective.

Remarks: Patient came to the hospital for diagnosis only. Was transferred home at the end of four days. Discharged, unimproved.

Case No. 31.—Medical Service.

M. F., female, age 40, married, U. S., admitted February 2,1921.

Past History: Negative.

Present Illness: Began two and one-half months ago with fever and night sweats, general muscular weakness, nervousness and dyspnoea, some loss of weight, also noticed oedema of ankles.

Physical Examination: Shows patient not acutely ill; teeth, many capped, otherwise negative; no lymphatic enlargement; lungs, few fine rales both bases posteriorly; heart, slight hypertrophy, blowing systolic at apex transmitted to left; pulse, regular; abdomen, central nervous system and extremities present no abnormalities. Urine is negative, Hb. 84%, erythocytes 4,600,00, leucocytes 6,000, polys. 61%. Blood chemistry including blood urea, creatinin, sugar and CO_2, all within normal limits. Blood pressure: systolic 110, diastolic 80. X-ray shows abscessed teeth with areas of rarefaction about several others. Blood culture was positive for Streptococcus viridans, on March 7, 1921, and persisted.

Clinical Course in Hospital: Patient was in the hospital for 68 days. The temperature for first sixteen days ranged around 99 to 101, pulse 110, respiration 25; during the remainder of her life it was higher, 100 to 104 with a pulse of 120, no change in respiration. Patient gradually lost weight and strength, pulse more feeble and, three weeks before she died, the temperature ranged around 100. Cardiac decompensation was a complication. The output of urine was always small as compared to the fluid intake, fair average ratio would be 30 to 90 oz.; albumin and casts a few weeks before her

death. The Hb. dropped to 55%, erythrocytes to 2,240,000, whereas the leucocytes rose to 13,800 with 81% polys.

Treatment: All carious teeth (abscessed) were removed, on February 18, 1921. Intravenous magnesium sulphate, 200 c.c., given on March 3, 1921; this had no effect on the pulse rate or temperature curve. One hour after injection of magnesium sulphate solution, patient had chill, temperature rose to 103, followed by profuse sweating; twenty-four hours later patient developed amnesia, could not think of words. Partial loss of pain and touch in right leg, with poor co-ordination in both; no motor paralysis. These disturbances gradually cleared up. Vaccines autogenous) $\frac{1}{2}$ c.c., commenced on March 10th and given for four weeks, every third or fourth day. The temperature became lower. Cardiac stimulation of tr. digitalis. Fluid intake was maintained mostly by Murphy's drip.

Diagnosis: Endocarditis, acute.

Remarks: Patient died. No autopsy.

Case No. 33.—Gyn. Service.

A. K., female, age 27, married, U. S., admitted March 31, 1921.

Past History: Negative.

Present Illness: March 22nd, patient had spontaneous miscarriage, about eight weeks pregnant; three days following, developed fever and vomited several times, some general abdominal pain and tenderness; four days later, patient developed joint pains, with swelling in small joints.

Physical Examination: General survey shows hands and wrists painful and swollen; heart and lungs are negative; indurated tender mass filling the pelvis (cul de sac), softened in spots; cervix is closed. Temperature 101.4, pulse 120, respiration 30. Urinalysis: Negative except for faint trace of albumin. Hb. 80%, leucocytes 27,800, polys. 87%. Blood culture positive for Streptococcus viridans, on April 5, 1921.

Clinical Course in Hospital: Patient was in the hospital fourteen days; temperature averaged 100 to 102 for eight days, when it gradually came to normal on the 11th day; nothing abnormal about pulse or respiration; April 19th, vaginal examination showed adnexa clear, slight mass posterior to uterus.

Treatment: Diet, soft, general, fluids were pushed; morphine

and codein for severe pain; ice bag to abdomen; sol. plumbi et opii to affected joints; (April 8th patient's condition much improved). Fowler position during acute infection; no operative interference.

Diagnosis: Salpingitis, subacute. Septicaemia, streptococcic.

Remarks: Infection was through the urogenital tract with transitory blood stream invasion; no metastatic infective foci. Discharged, recovered.

Case No. 40.—Medical Service.

D. F., female, age 31, single, nurse, U. S., admitted January 29, 1922.

Past History: Heart trouble for past eight years, with dyspnoea at times. Admitted to Brooklyn Hospital October 18, 1921, with digestive symptoms; her appendix and gall-bladder were removed, both showed low grade inflammatory changes. She had cardiac hypertrophy and a blowing systolic murmur at apex at this time.

Present Illness: In her usual health until one month ago when she began to feel worse; afternoon temperature was 102 to 103; complained of headache, pains in the extremities, palpitation, and noticed reddish spots appearing on her extremities.

Physical Examination: General survey shows an undernourished female with skin and mucous membranes pale; petechial spots present on arms and toes; teeth and tonsils appear in good condition. Lungs—Few fine rales over both bases behind, with impaired resonance. Heart is hypertrophied to the left, systolic murmur at the apex and transmitted to left. Abdomen shows liver normal in size, spleen enlarged to free costal border. Extremities, negative. Urinalysis: 1200 c.c. in 24 hours; sp. gr.. 1010; trace of albumin; no sugar; few hyaline and granular casts and red blood cells. Hb. 60%, erythocytes 4,280,000, leucocytes 10,200, polys. 80%. X-ray of teeth shows small abscess at root of left lower lateral incisor. Blood culture positive for Streptococcus viridans, on February 3, 1922.

Clinical Course in Hospital: Patient remained in the hospital 63 days. Her temperature varied between 98-100 in the A.M. to 101-103 for P.M., pulse averaged 110, respirations 30 per minute. During the entire period of observation, she complained of chilly feelings much of the time with sweating and definite muscular and

joint pains. On March 15, 1922, the Hb. had dropped to 43%, the red cells to 1,640,000, and the leucocytes to 8,900. On March 31, 1922, she suddenly developed paralysis of right arm, leg and face, due to cerebral embolism, and died twenty-four hours later.

Treatment: Full diet. Autogenous vaccines every third day at first and later every fifth day throughout the stay in hospital. Zambeletti's arsnic and iron solution by hypo. Codein and morphine to relieve restlessness.

Diagnosis: Subacute bacterial endocarditis with complication of cerebral embolism.

Remarks: Portal of entry unknown. Patient died.

Streptococcus Hemolyticus Infections.

Case No. 6.—Medical Service.

J. R., male, age 48, married, Russia, admitted May 18, 1917.

Past History: Removal of calculus in kidney six years ago. Denies venereal disease.

Present Illness: Well until three months ago when he suffered from a peritonsillar abscess, lasting two weeks; apparently entirely recovered when three weeks later again suffered an attack of acute tonsillitis, this time associated with general multiple arthritis, shoulders, wrists, knees, and ankles; these symptoms have never entirely cleared up; last ten days became much worse with arthritic trouble and in addition has complained of chilly feelings, fever, followed by profuse sweating.

Physical Examination: General survey shows patient subacutely ill; shoulders, wrists, elbows, and ankles swollen, red, and tender. Lungs, negative. Heart is not hypertrophied, no murmurs, rapid. Abdomen—Liver and spleen are normal, no masses or tenderness. Urinalysis: Trace of albumin, granular casts. Temperature 101, pulse 120, respirations 30. Leucocytes 12,500, polys. 69%. Blood culture positive for Streptococcus hemolyticus, on May 21, 1917.

Clinical Course in Hospital: Patient was in hospital 24 days, during which time he made a gradual recovery; temperature reaching normal on the seventh day.

Treatment: Soft farinaceous diet, fluids pushed. Initial dose

of calomel and soda given, followed by magnesium sulphate each A.M. for three days. Sodii salicylate and bicarbonate soda of each 20 gr. q. 2 h. for 48 hours, after this time it was given t.i.d.. Oil gaulheria m iv q. 3h. for 10 days. Argyrol spray, 205 sol., to throat q.d. for the first week.

Diagnosis: Rheumatism, acute articular.

Remarks: Portal of entry probably tonsils. Discharged, recovered.

Case No. 8.—Gyn. Service.

L. F., female, age 31, married, Norway, admitted May 29, 1918.

Past History: Negative.

Present Illness: Three weeks ago had an induced abortion, at about two months pregnant. Two weeks ago she became ill with fever, difficulty in breathing, and, one week ago, wrists became red, swollen and tender.

Physical Examination: General survey is negative; heart shows no murmurs; lungs are negative; no petechial areas in skin. Pelvic examination reveals mass in pelvis, probably an abscess. Urinalysis: Trace of albumin, otherwise negative. Temperature 104, pulse 140, respirations 35 per minute. Hb. 62%, leucocytes 7,000, polys. 80%. Blood culture positive for Streptococcus hemolyticus, on May 31, 1918.

Clinical Course in Hospital: Patient was in the hospital 123 days. On June 10, 1918, the pelvic abscess was opened and drained from below, considerable foul smelling pus removed. Her temperature fluctuated betwen 98 and 102 with pulse around 128, until June 23rd when it reached the normal mark. On June 24, 1918, she developed swelling and tenderness on dorsum of right foot, left wrist slightly improved; heart sounds are of poor quality but no evidence of endocarditis. During the patient's stay in hospital she developed an abscess on dorsum of left hand, another on right foot and on thigh, all of which needed incision and drainage. One month after admission she developed signs of low grade pelvic peritonitis, which later drained through the umbilicus. She developed superficial abscesses and suffered from multiple arthritic lesions but did not show any deep infective foci in the internal organs.

Treatment: Nutritious diet, fluids pushed. On June 2, 1918, patient was given intravenously 150 c.c. of 3% magnesium sulphate sol.; some reaction followed, sweating freely. On June 10, 1918, incision and drainage of pelvic abscess from below. Mixed stock vaccine for several weeks.

Diagnosis: Abortion, pelvic abscess. Septicemia.

Remarks: Discharged, improved. Social Service reports that patient is in excellent health, on April 15, 1922.

Case No. 39.—Gyn. Service.

M. S., female, age 28, married, U. S., admitted May 16, 1920.

Past History: Negative.

Present Illness: Patient was five months pregnant when abortion was induced, sixteen days before admission to the hospital. Soon after the abortion she developed fever; for the last three days the patient has been much worse; complained of abdominal tenderness and distention, vomiting on several occasions and constipation.

Physical Examination: General survey shows patient critically ill, she is irrational at times; profuse vaginal discharge; lungs negative; heart negative; abdomen greatly distended and tympanetic, very little peristalsis; extremities negative. Vaginal examination shows no masses. Blood culture positive for Streptococcus hemolyticus, on May 17, 1920.

Clinical Course in Hospital: Patient was in the hospital four days. The temperature, on admission, was 97, pulse 120, respiration 28, and her condition was so poor that, by local abdominal anesthesia, a right rectus incision was made and a large amount of free abdominal pus was found; omentum in places was gangrenous, it was ligated and removed and rubber tube drainage established. Her condition did not improve, the temperature remained between subnormal and 99 until just before death, when it reached 105.

Treatment: Application of external heat; cardiac stimulants; colonic irrigation; pituitrin by hypo. for abdominal distention.

Diagnosis: Peritonitis, acute, suppurative. Abortion, induction of, before Streptococcic septicemia.

Remarks: Patient died. Autopsy showed uterus corresponding in size to about a two months pregnancy. Lower half of abdomen

involved in a diffuse suppurative peritonitis and extending on the right side up to the liver.

Streptococcus Brevis Infections.

Case No. 4.—Medical Service.

F. P., female, age 22, married, Roumania, admitted April 18, 1917.

Past History: Measles as child; dyspnoea for last eight years, no cough; no severe illness; one child, two years of age.

Present Illness: In her usual health until three weeks ago when she contracted a "heavy cold"; complained of malaise, cough, muscular weakness, chilly feelings and sweating for last two days.

Physical Examination: General survey shows patient acutely ill, face flushed and lips are slightly cyanotic; teeth are fairly well kept; tonsils and pharynx negative; pulsating carotids; skin clear. Lungs are negative. Heart is markedly hypertrophied, apex beat in sixth interspace 12 cm. from midsternal line, heaving in character; there is a presystolic thrill over mitral area; at the apex a short presystolic murmur, not transmitted, and a systolic transmitted into axilla; there is a double murmur at aortic area, the diastolic transmitted across and down the left of the sternum. Abdomen—Liver and spleen of normal size. Extremities not oedematous. Pulse, water-hammer in character, regular. Temperature 102, pulse 110, respirations 30 per minute. Urinalysis: Negative except for trace of albumin. Blood pressure: Systolic 150, diastolic 60. Leucocytes 21,400, polys. 81%. Wassermann: Negative. Blood culture positive for Streptococcus brevis, on April 25, 1917.

Clinical Course in Hospital: Patient was in hospital thirty-five days. The P.M. temperature ranged around 104 for seven days, when it dropped to 102 for ten days, the A.M. temperature being 99, reaching normal on the thirty-first day in hospital; the pulse varied from 90 to 120 in rate, respiration always a trifle rapid. There were no signs of any complications while in the hospital. The partial cardiac decompensation was always in evidence.

Treatment: Soft diet, milk and gruels especially. Tr. digitalis as cardiac support. Patient out of doors on porch as much as possible. Magnesium sulphate sol. 250 c.c. introduced into the veins,

no reaction following the injection but the temperature began to fall and gradually came to normal, seven days after the injection.

Diagnosis: Acute endocarditis. Chronic valvular heart disease.

Remarks: Portal of entry not known, unless it was through the respiratory tract. Discharge, improved. Social Service reports that patient died September 1920.

Case No. 5.—Medical Service.

L. R., male, age 44, married, candy manufacturer, U. S., admitted May 16, 1917.

Past History: Attack of rheumatism one year ago. Denies venereal disease.

Present Illness: Well until nine days ago when he complained of pain in right shoulder, this shifted to his ankle and knee. Yesterday, moderate pain in both wrists. No respiratory, cardio-vascular, or digestive symptoms.

Physical Examination: General survey shows patient subacutely ill; no eruption; pharynx and tonsils moderately injected; lymphatics at angle of jaw tender; teeth sound. Lungs negative. Heart not hypertrophied, no murmurs, sounds of poor muscular quality. Abdomen—Liver and spleen normal, no masses or tenderness. Wrists moderately swollen and tender, right shoulder tender, likewise both knees, elbows stiff. Urinalysis: Negative. Temperature 102, pulse 100, respirations 30 per minute. Blood pressure: Systolic 140, diastolic 80. Leucocytes 16,600, polys. 83%, Hb. 84%, erythocytes 4,950,000. Wassermann: Negative. Blood culture positive for Streptococcus brevis, on May 18, 1917.

Clinical Course in Hospital: Patient remained in hospital thirty-one days. The temperature fluctuated between 102 in P.M. and 99 in A.M. for eleven days after admission, when it gradually became normal on the seventeenth day; the pulse and respirations were within normal limits. There were no complications.

Treatment: Diet of general fluids. Sodii salicylate, sodii bicarbonate. Magnesium sulphate, 150 c.c. intravenously, was given to the patient on May 19th and repeated on May 26th; no reaction noticed following either injection, the temperature gradually falling to normal soon after the last injection.

Diagnosis: Acute articular rheumatism. Streptococcic septicaemia.

Remarks: Patient showed no complications. Portal of entry naso-pharynx. Discharged, recovered.

Case No. 7.—Medical Service.

E. G., female, age 17, single, clerk, U. S., admitted June 4, 1917.

Past History: Negative.

Present Illness: Well until two weeks ago, when patient began with general malaise associated with increasing muscular weakness requiring her to go to bed; no cough; no digestive disturbances. On May 29, 1917, after getting out of bed and going out of doors, she was seized with a chill followed by fever and dizziness, vomited once; no diarrhea; no sore throat; no headache; no epistaxis.

Physical Examination: General survey reveals young girl acutely and seriously ill, in state of profound toxaemia; skin clear; no cerebro-spinal signs; tongue and lips parched, and small hemorrhagic area in mucous surface of mouth; oral cavity intensely congested; heart rapid, no murmurs, weak sounds; lungs show few musical rales posteriorly but no sign of consolidation; abdomen distended, liver and spleen of normal size, no masses or points of tenderness; extremities, negative; knee-jerks, active. Urinalysis: Heavy trace of albumin, but no casts. Temperature 104, pulse 130, respirations 35 per minute. Blood pressure: Systolic 100, diastolic 70. Leucocytes 12,000, polys. 71%. Blood culture was positive for Streptococcus brevis, on June 7, 1917.

Clinical Course in Hospital: Patient was in hospital four days, during which time she showed increasing signs of septic poisoning. The urine showed large amount of albumin without casts or red blood cells. At times she was delirious and in profuse perspiration. Patient showed increasing prostration and stupor and died on the fourth day in hospital.

Treatment: Diet of general fluids; cardiac stimulation; colonic irrigations; turpentine stupe for ileus.

Diagnosis: General streptococcic septicaemia.

Remarks: The patient died of general sepsis. No autopsy allowed.

Case No. 22.—Medical Service.

J. G., female, age 40, married, admitted December 8, 1919.

Past History: Subject to sore throats.

Present Illness: Well as usual until November 27, 1919, when she suffered from an attack of acute tonsillitis. Soon after the tonsillitis she complained of chills followed by fever; associated with these symptoms a diffuse swelling appeared involving left cheek and neck, which has steadily increased in size. No respiratory or gastro-intestinal symptoms.

Physical Examination: General survey reveals patient bathed in perspiration but not suffering much discomfort; skin anaemic and has waxy appearance; reflexes normal, no signs of meningial irritation; left side of cheek and neck markedly swollen and indurated, no fluctuation; left shoulder joint tender to motion, likewise left hip; no enlarged lymphatic glands; skin is clear, save for over-swelling in neck; lungs are negative; heart not hypertrophied, soft blowing systolic at apex; throat reddened but no exudate; cerebro-spinal nervous system negative. Urinalysis: Negative save for faint trace of albumin. Temperature 102.8, pulse 120, respiration 40. Leucocytes 19,800, polys. 90%. Blood culture positive for Streptococcus brevis, on December 10, 1919.

Clinical Course in Hospital: Temperature remained high, 102 to 105, pulse averaged 120 to 150 per minute, respirations crept up from 30, on admission, to 45 on fourth day in hospital. The patient became more septic and died on the fifth day after admission. The cellulitis of neck and arthritis of shoulder and hip unchanged.

Treatment: Surgical consultation on December 8, 1919, by Dr. Sherwood, confirmed the diagnosis previously made and advised no surgical interference at present. Diet was general fluids; catharsis; cardiac stimulants as indicated; wet dressing to cellulitis in neck; December 9th antistreptococci serum was given intravenously, 100 c.c., no reaction; December 10th 50 c.c. of serum given, no reaction; six hours later again 50 c.c. were given, and six hours later the patient seemed brighter and condition somewhat improved; December 11th, twelve hours after last intravenous of serum, 50 c.c. more were given and repeated again in six hours; Murphy's drip of

glucose solution was used at intervals; saline intravenously was used on two occaions.

Diagnosis: Acute septic endocarditis. Cellulitis of neck.

Remarks: Patient died on fifth day after admission to hospital. No autopsy.

Staphylococcus Aureus Infections.

Case No. 3.—Medical and Surgical Services.

J. C., male, age 57, married, motorman, U. S., admitted March 14, 1917.

Past History: Venereal disease denied, good health.

Present Illness: For past two years has noticed tumor, steadily growing in size, situated over upper sternum; has not given him any discomfort. Has complained of "bronchitis" for past three weeks; cough, fever, no night sweats.

Physical Examination: General survey shows man only fairly nourished, slightly cyanotic, skin dry, naso-pharynx congested, tongue coated. There is a round mass three inches in diameter and raised three-quarter inch above surrounding parts; the tumor is reddened in places and gives distinct fluctuation; skin is not apparently broken over the mass. Both lungs, posteriorly, show many fine moist rales. Heart not hypertrophied, no murmurs. Rest of body is negative. Urinalysis: Negative. Blood pressure: Systolic 150, diastolic 100. Leucocytes 7,900, polys. 72%. Wassermann: Negative. Blood culture positive for Staphylococcus aureus, on May 25, 1917.

Clinical Course in Hospital: Patient was in hospital sixteen days, during which time his temperature averaged around 101 to 102 in the P.M., with normal temperature in the A.M., for the first five days; at this time the tumor was aspirated and bloody pus removed, this material was negative for acid fast bacilli but unfortunately no other bacteriological examination was made. The temperature curve remained the same. On account of the positive blood culture, the patient was given nitrous oxid gas and the sternal mass removed; in three days his temperature dropped to normal. The pathological report on tissue removed was that the tumor is an infectious granuloma (probably syphilitic).

Treatment: Removal of tumor surgically.

Diagnosis: Gumma of sternum, infected.

Remarks: Portal of entry probably through infected gumma. Discharged, recovered.

Case No. 20.—Pediatric Service.

S. DeC., male, age 8, school, U. S., admitted August 27, 1919.

Past History: Convulsions five years ago.

Present Illness: Was in good health until fifteen days ago when he complained of pain and swelling over dorsum of both feet; no history of tonsillitis, skin lesions, respiratory or gastro-intestinal symptoms; twenty-four hours before admission he developed marked dyspnoea and temperature was higher, no chills.

Physical Examination: Reveals boy "in extremis," partially comatose, pale, markedly dyspnoeic, face pinched, tongue heavily coated; lungs, negative; heart, rapid and thready, some enlargement to left, no murmurs; dorsum of both feet and back of left hand including wrist, red, tense, swollen with fluctuation; some disintegration of fifth tarso-metatarsal joint of left foot; no cerebro-spinal symptoms. Temperature 104, pulse 140, respirations 50 per minute. Blood culture positive for Staphylococcus aureus, on August 28, 1921.

Clinical Course in Hospital: Fluctuating masses on feet were aspirated and cultured, with result that Staphylococcus aureus was found in pus aspirated. Patient remained in hospital forty hours, when he died.

Treatment: Incision and drainage; free cardiac stimulation; Murphy drip.

Diagnosis: Acute arthritis, multiple. Septicaemia, staphylococcic.

Remarks: Portal of entry, undetermined. Patient died. No autopsy allowed.

Case No. 25.—Gyn. Service.

G. L., female, age 30, married, Ireland, admitted May 30, 1920.

Past History: Negative as to general health; five children, one miscarriage at three months, about two weeks ago; no constitutional symptoms.

Present Illness: Entered hospital for repair of pelvic floor.

Physical Examination: General survey shows healthy woman; heart, lungs, extremities negative; some general abdominal tenderness, no masses felt; slight brownish vaginal discharge; temperature 98.8, pulse 90, respiration 20. Urinalysis: Negative save for faint trace of albumin. Blood pressure: Systolic 110, diastolic 60. Blood culture positive for Streptococcus aureus, on June 14, 1920.

Clinical Course in Hospital: Patient was in the hospital sixteen days. She was observed for nine days, at which time she was given a general anesthetic and repair of pelvic floor was done. Three days after operation her temperature rose to 102, pulse 110, respiration 20, leucocytes 10,600, polys. 78%, erythrocytes 4,500,000. The perineum was reddened and there was evidence of a pelvic cellulitis, tenderness over lower quadrant of abdomen. The temperature and pulse continued to rise, the highest temperature being 106, which was just before she died—death being the result of sepsis secondary to pelvic cellulitis.

Treatment: Pelvic drainage was instituted through the wound of operation; urotropin and cardiac stimulants were given following the rise in temperature; fluids were pushed; bicarbonate of soda 5% sol. given per rectum.

Diagnosis: Pelvic cellulitic. Septic broncho-pneumonia.

Remarks: Patient had infection in wound of operation, and sepsis followed; patient died four days after. Portal of entry: Perineal operative wound. Patient died. No autopsy.

Case No. 27.—Medical Service.

A. F., male, age 15, single, school, Italian, admitted September 22, 1920.

Past History: Pneumonia, left, in 1917; sero-fibirnous pleurisy, left, in 1918, aspirated and fluid removed; otherwise has enjoyed good health.

Present Illness: Began following an attack of pleurisy in 1918; suffered more or less pain in left chest and cough with expectoration at times. Last six months lost ten pounds, and since September 15, 1920 pain in chest has been worse with a tight feeling associated with

chilliness followed by fever. The sputum, moderate in amount, is greenish in color and at times blood tinged.

Physical Examination: General survey shows boy emaciated; skin is dry; fingers a trifle clubbed and hands cyanotic, especially on exercise; tonsils enlarged, teeth good; left chest measure 35 cm., right 37.5 cm.; left lung is more or less fixed, especially lower half; physical signs in left lower chest of thickened pleura with an area of distinct bronchial breathing over the fourth to eighth intercostal spaces posteriorly, with many medium and fine moist rales; right lung shows evidence of an associated bronchitis, apices are clear. Heart is not hypertrophied, slight systolic at apex, pulmonic 2nd greater than aortic 2nd. Abdomen—Liver and spleen or normal size, no masses or points of tenderness. No abnormalities present in cerebro-spinal tract. Temperature 99.5, pulse 120, respirations 35 per minute. Urinalysis: Negative. Blood pressure: Systolic 90, diastolic 50. Sputum negative for acid fast bacilli. Leucocytes 11,000, polys. 70%, Hb. 65%. Von Pirquet's reaction for tuberculosis is negative. Wassermann: Negative. X-ray shows blocking of the rays in left lower chest and includes the left costo-diaphragmatic angle; this is a dense circular shadow about 4 cm. in diameter just to the left of the heart shadow, suggestive of lung abscess with sero-fibrinous pleurisy. Blood culture positive for Staphylococcus aureus, on October 2, 1920.

Clinical Course in Hospital: Patient was in the hospital forty days. The temperature, from his admission until October 20, averaged 101 for P.M. and 99 for A.M., pulse 110, respiration 30 per minute. The left chest was aspirated twice during this period with negative results. On October 20th, his condition not having improved, he was given a local anesthetic of novocain, blocking off the left 6th, 7th, 8th intercostal spaces posteriorly; aspiration in the 8th space in post axillary line was done and pus of foul odor aspirated, followed by incision and drainage. Culture of the pus aspirated was sterile; microscopic examination of debritis removed showed only necrotic material. At the time of opening the abscess cavity patient expectorated frothy, purulent, bloody material, evidently through a bronchial fistula. Temperature dropped to normal on the fourth day following drainage. On November 16, 1920, he suffered

from insufficient drainage of abscess cavity and after enlarging the drain opening in the chest wall, temperature again fell to normal.

Treatment: General high caloric diet; out of doors on porch much of the time; mixed vaccine, 1 c.c. every fourth day, for a period of four weeks was given hypodermatically.

Diagnosis: Pulmonary abscess, left. Pleurisy, chronic fibrinous.

Remarks: On December 29, 1920, moderate amount of drainage from fistula in left chest; general condition, fair. Discharged, improved, on December 5, 1920.

Case No. 32.—Surgical Service.

E. H., male, age 40, married, waiter, U. S., admitted March 3, 1921.

Past History: Lues nine years ago. "Water on the knee," twelve years ago.

Present Illness: Two weeks ago wrenched his left knee, after which it became painful and swollen.

Physical Examination: Shows teeth, many carious; heart, lungs and abdomen are negative; left knee measures in circumference 40 cm., right knee 35 cm., no fluid; tissues about knee joint thickened and inflamed. Temperature 99.4, pulse 90, respiration 20. Urinalysis: Negative. Wassermann: Four plus. Blood culture positive for Staphylococcus aureus, on April 1, 1921.

Clinical Course in Hospital: Patient remained in the hospital thirty-four days. Left knee was aspirated on March 4th, joint irrigated with normal saline solution; cultures made of aspirated fluid were sterile. Temperature ranged around 101 for five days following the surgical interference, when it dropped to normal and remained so until the day of discharge of the patient from the hospital.

Diagnosis: Arthritis, subacute, left knee.

Remarks: Discharged, improved.

Staphylococcus Albus Infections.

Case No. 34.—Medical Service.

H. W., female, age 21, married, U. S., admitted May 11, 1917.

Past History: Negative.

Present Illness: Enjoyed good health until May 3, 1917, when she was seized with a severe chill, which was followed by fever and sweating; complained also of aching in her back and extremities, throat was sore; in a few days her joints became stiff and painful especially the elbows, gradually getting worse since onset of trouble; no cerebro-spinal or digestive disturbances.

Physical Examination: General survey reveals woman acutely ill, but well nourished; anxious expression; schlera slightly jaundiced; many carious teeth and poorly kept; pharynx moderately congested, tonsils the same; skin is clear; lungs are negative; heart is negative for hypertrophy and murmurs; abdomen—liver and spleen or normal size, there is slight distension and moderate tenderness in left lower quadrant, no masses felt; extremities are negative. Temperature 103, pulse 130, respirations 25 per minute. Urinalysis: Sp. gr. 1025, acid, trace of albumin, no sugar, moderate number of pus cells, few hyaline casts and red blood cells. Leucocytes 3,600, polys. 78%; another count in six hours reveals 3,500 leucocytes. Blood culture was positive for Staphylococcus albus, on May 12, 1917.

Clinical Course in Hospital: Soon after the temperature rose to 105, pulse 130, respiration 50. The skin became slightly jaundiced, and pus with red blood cells was found in the urine. She rapidly became more septic and died forty-eight hours after admission to the hospital.

Treatment: Diet of general fluids; calomel and magnesium sulphate as a cathartic; cardiac stimulants; sodii bicarbonate, solution 5%, as drink.

Diagnosis: Septicemia, general, due to Staphylococcus albus.

Remarks: Portals of entry probably the tonsils. Patient died.

Autopsy, May 13, 1917, revealed a Staphylococcus albus septicemia with metastatic abscess in righ kidney; petechial areas over the visceral, pericardial, and endocardial surfaces. Liver simply showed a toxic hepititis; microscopic section of kidney revealed masses of Staphylococci about the abscess cavities. The heart was taken under asceptic precautions and revealed a pure culture of Staphylococcus albus.

Bacillus Coli Communis.

Case No. 36.—Surgical Service.

G. T., male, age 29, married, mechanic, U. S., admitted October 16, 1921.

Past History: Irrevalent.

Present Illness: In August 1921, patient had attack of right sided abdominal pain, especially over gall-bladder area; this has continued with more or less severity.

Physical Examination: General survey shows man, well developed and nourished; heart and lungs are normal; abdomen and extremities, negative. Urinalysis: Negative, three consecutive specimens, each on a different day, reveal no acid fast bacilli. X-ray is negative for renal or ureteral calculi. Gastro-intestinal Barium Series, negative except for appendix shadow.

Clinical Course in Hospital: Patient remained in hospital twenty-one days. On October 17, 1921, patient was given general anaesthetic and gall-bladder and appendix were removed; microscopic examination of both of these organs showed chronic cholecystitis and chronic appendicitis; cultures taken at time of operation from gall-bladder reveal bacillus coli communis and bacillus aerogenes capsulatus. Temperature was intermittant from time of operation until day of discharge, the variation being between 98 and 101. Patient left the hospital November 6, 1921, gall-bladder wound open and draining.

Readmitted on November 13, 1921, with temperature 102, pulse 118, respirations 25 per minute; complaining of general weakness and vomiting of bile stained material.

Physical Examination: Shows patient prostrated; skin of dusky pallor; sclera shows icterus; phlegmon in region of left parotid. Urinalysis: Sp. gr. 1012, acid, trace of albumin, few red blood cells, many pus cells. Leucocytes 15,000, polys. 75%. Cystopic examination, on December 19, showed urine normal from right kidney; few small clumps of pus cells, moderate number of red blood cells from left kidney. Cultures from left kidney were postive for B. Coli communis, right kidney, sterile. Blood culture was positive for Bacillus coli communis, on November 16, 1921.

Clinical Course in Hospital: The patient was in the hospital

forty-one days during his readmission. The temperature gradually becoming normal on the thirty-eighth day. On November 23rd, was given nitrous oxide gas; incision and drainage in parotid tumor, pus evacuated. B. Coli vaccine was started and repeated every three days at first, then once a week until December 11, 1921. The coli sepsis gradually cleared up without any complicating lesions.

Treatment: The diet was high caloric, fluids and soft, at first and later. The cardio-vascular system needed practically no medicine.

Diagnosis: Pyelitis, B. coli infection.

Remarks: Portal of entry: Gastro-intestinal tract. Discharged, improved.

Follow up Notation: Report of Dr. Rathbun, on April 10, 1922, is that the left kidney shows, at this date, apylonephrosis.

Case No. 38.—Surgical Service.

T. A., male, age 42, married, Sweden, admitted December 10, 1921.

Past History: Negative.

Present Illness: For three days complained of colicky pains in abdomen, becoming localized over gall-bladder region; bowels constipated; vomited on several occasions, a sour material. Pain so severe that morphine was given previous to hospital care; he has had no relief from his pain. No complain of chills, cough, or jaundice.

Physical Examination: General survey reveals middle aged man, well developed and nourished, acutely ill and suffering severe abdominal pain; tongue markedly coated; impaired resonance and distant breath sounds over right lower chest; heart lacks muscular tone, otherwise is negative; abdomen shows general rigidity, more so in right upper quadrant where there is marked tenderness, dull percussion note over entire abdomen. Blood pressure: Systolic 100, diastolic 80. Blood culture positive for B. Coli communis, on December 12, 1921.

Clinical Course in Hospital: Patient remained in the hospital seven days, during which time the temperature ranged about 101 or 102, pulse 130, respirations 25 to 30. Immediately after admission, a right incision was made, gall-bladder removed and wound drained.

The peritoneal cavity contained a large amount of foul smelling fluid, gall-bladder was gangrenous, no calculus, some exudate on the bowel wall. He gradually became more septic and died, seven days after operation.

Treatment: Diet of general fluids; Murphy drip; saline hypodermoclysis given at intervals; morphine sulphate for abdominal pain; pituitrin for abdominal distention; cardiac stimulation consisted of camphor in oil, caffein et sodii benz.; digifoline by hypo.

Diagnosis: Cholecystitis, gangrenous. Peritonitis, general.

Remarks: Patient died. Autopsy revealed: Acute general sero-fibrinous peritonitis, secondary to gall-bladder disease. Bronchopneumonia. Chronic pleurisy, right. Microscopic examinations were made of lungs, peritoneum,gall-bladder and kidney.

Pneumococcus Type II, III, IV Infections.

Case No. 9.—Medical Service.

S. S., male, age 18, single, West Indies, admitted October 1, 1918.

Past History: Negative.

Present Illness: Well until five days ago when he complained of cough, headache, fever; sputum was blood stained; bowels constipated; considerable prostration; no chills.

Physical Examination: General survey reveals patient acutely ill; respiration, labored. Lungs—Signs of consolidation at both lower lobes and right upper anteriorly, no pleural involvement. Heart rapid, otherwise negative. Remainder of body is negative. Urinalysis: Negative. Temperature 104, pulse 120, respirations 30 per minute. Blood pressure: Systolic 100, diastolic 50. Leucocytes 18,000, polys. 80%. Sputum positive for Pneumococcus, type not determined. Blood culture positive for Pneumococcus type II, on October 2, 1918.

Clinical Course in Hospital: Patient was in the hospital five days during which time his temperature continued high, 102 to 105, until the day of his death, when the temperature began to fall; pulse and respiration were proportionate to the temperature.

Treatment: Diet, general fluids; cathartic of castor oil on admission, followed by daily enema; influenza vaccine (stock) was commenced at time of admission; free cardiac stimulation of

digitalis, camphor, caffein et sodii benzoate; alcohol sponges for high temperature.

Diagnosis: Influenza. Pneumonia type II, double lobar.

Remarks: Portal of entry—Respiratory tract. No complications. Patient died from extensive lung involvement plus high grade toxemia.

Case No. 10.—Medical Service.

J. S., male, age 33, married, electrician, U. S., admitted January 13, 1919.

Past History: Negative.

Present Illness: Well until twenty-four hours ago when he was seized with severe headache, chest pains and cough, some fever; no chill; no digestive symptoms.

Physical Examination: General survey reveals patient acutely ill; patchy area of crepitant rales; posteriorly on both sides; heart regular, no murmurs; remainder of body normal. Urinalysis: Negative. Temperature 103, pulse 120, respirations 25 per minute. Sputum shows Pneumococcus type III. CO combining power of blood 34.7. Blood culture was positive for Pneumococcus type III, on January 22, 1919.

Clinical Course in Hospital: Patient was in the hospital for twenty-eight days. On January 17th he had an extension of the pneumonia to the left upper lobe, with rise in temperature, pulse and respiration, returning to normal on January 25th. Soon the temperature began to be irregular, showing an afternoon temperature of 102. The pulse during this stormy course became irregular and varied between 80 and 130 per minute; the respirations reaching as high as 50 per minute some days. The physical signs in the chest showed a migrating broncho-pneumonia with considerable involvement of the pleura; paracentesis was done but no signs of pus until on February 8th, the day before he died, purulent fluid was aspirated from his right chest. No cerebro-spinal symptoms.

Treatment: High caloric soft diet was given him throughout his illness; cardiac stimulation of digitalis, camphor, caffein et sodii benzoate; morphine sulphate and codein together with mustard paste for the relief of pain; Murphy drip of 10% bicarbonate of soda or 5% glucose solution was resorted to as the oral intake dropped off.

Diagnosis: Influenza. Broncho-pneumonia type III.

Remarks: Complication, empyema. This patient showed a virulent infection from the onset, as the blood and the sputum showed Pneumococcus type III his chances of recovery were, from the beginning, poor. The pneumonic lesion contra-indicated opening the chest at the time pus was found. The following day he showed signs of sudden cardiac failure and died. No autopsy.

Case No. 11.—Medical Service.

J. M., male, age 30, married, pattern maker, U. S., admitted January 23, 1919.

Past History: Negative.

Present Illness: Suddenly, three days ago, began to complain headache, chest pains, and dry cough. These symptoms gradually became worse and fever developed with considerable prostration; no digestive or nervous symptoms.

Physical Examination: General survey reveals patient seriously ill, general cyanosis, pulse weak. Lungs—Show dullness; areas of bronchial breathing and many crepitant rales over both lungs posteriorly; no evidence of pleural involvement. Heart—Irregular, sounds of poor quality, no murmurs. Abdomen and extremities are negative. No urinalysis was made. Temperature 104, pulse 130, respirations 40 per minute. No leucocyte count was made. Blood culture was positive for Pneumococcus type III, on January 23, 1919.

Clinical Course in Hospital: Patient was in the hospital less than twenty-four hours when he died from profound sepsis.

Treatment: Diet of general fluids which were pushed; castor oil as cathartic; free cardiac stimulation; alcohol sponges for the high temperature and delirium.

Diagnosis: Influenza. Pneumonia, double lobar.

Remarks: Patient died. No autopsy allowed.

Case No. 12.—Medical Service.

M. R., female, age 24, single, child's nurse, Ireland, admitted January 24, 1919.

Past History: Always enjoyed good health.

Present Illness: Suddenly, three days ago, began to complain

of slight headache with general malaise. Patient says she does not feel acutely ill.

Physical Examination: General survey is normal, save for respiration of 30, pulse 90, temperature 100; heart is negative; lungs, no abnormal signs present; abdomen is negative. Urinalysis: Negative. Blood pressure: Systolic 100, diastolic 40. Blood culture positive for Pneumococcus type IV, on January 31, 1919.

Clinical Course in Hospital: Patient was in the hospital three days before there were true diagnostic signs, when her temperature rose to 105, pulse 100, respiration 35. Gastro-intestinal symptoms developed as pain and nausea with difficulty in breathing and extreme exhaustion; the skin is somewhat blanched. The lungs at this time revealed signs of a broncho-pneumonia, especially on the right side; this rapidly extended in twenty-four hours so that definite physical signs of a massive broncho-pneumonia could be made out; the temperature remained around 104 to 105, pulse became irregular and more rapid, the respiration increased to 40, the CO combining power of the blood 36.1, and the patient died, on the eighth day after admission, from an overwhelming toxemia.

Treatment: Influenza precautions. Soft diet, force fluids; cardiac stimulation of digitalis, camphor in oil; bicarbonate of soda by mouth and rectum; strychnine sulphate; caffein et sodii benzoate.

Diagnosis: Influenza. Broncho-pneumonia, massive.

Remarks: Patient entered hospital not acutely ill, with few physical signs, when, on the third day, she developed a massive broncho-pneumonia with marked toxemia, especially affecting the neuro-vascular system, and died. Patient died. No autopsy allowed.

Case No. 13.—Medical Service.

M. T., male, age 29, married, shoemaker, Italy, admitted January 25, 1919.,

Past History Negative.

Present Illness: Well until nine days ago when he was seized with severe aching of the body, cough, fever, and headache; somewhat constipated and twitched at onset; chest pains severe for last few days.

Physical Examination: General survey shows patient acutely ill, no cyanosis, pulse not rapid but of poor quality, tongue coated,

breathing rapidly. Heart is negative. Lungs show impaired resonance both bases posteriorly, with many fine moist rales and patchy broncho-vesicular breathing. Urinalysis: Negative. Temperature 103, pulse 100, respirations 30 per minute. Blood pressure: Systolic 110, diastolic 40. Sputum positive for Pneumococcus type III. Blood culture positive for Penumococcus type IV, on January 28, 1918.

Clinical Course in Hospital: Patient was in the hospital thirteen days, entering on the tenth day of his disease. A fibrinous pleurisy was manifest at left base forty hours after admission . Temperature remained high for three days when it returned to normal, pulse and respiration reaching normal on same day.

Treatment: Diet of general fluids; routine digitalis; open air on suitable days; alcohol sponges for high temperature.

Diagnosis: Influenza. Broncho-pneumonia; developed acute fibrinous pleurisy as a complication.

Remarks: Patient required very little cardiac stimulation and received few drugs. Open air treatment was fairly constant. Discharged, recovered.

Case No. 14.—Medical Service.

G. J., male, age 27, single, printer, S. A., admitted January 26, 1919.

Past History: Negative, always well and healthy.

Present Illness: Well until eight days ago when he developed fever, headache and cough, worse the last three days.

Physical Examination: General survey is negative; heart normal; lungs show many scattered fine crepitant rales over both bases, with impairment of resonance on right base posteriorly, no change in breath sounds; some general lagging in the respiratory excursion. Urinalysis: Negative. Temperature 103, pulse 108, respiration 28. Blood pressure: Systolic 110, diastolic 60. Sputum positive for Pneumococcus type IV. Blood culture positive for Pneumococcus type IV, on January 31, 1919.

Clinical Course in Hospital: Patient remained in hospital twenty-one days; temperature reached normal on the seventh day

by lysis, the pulse and respiration likewise returned to normal on the same day, at which time the physical signs had disappeared.

Treatment: Consisted of the usual isolation and soft diet during the active infection; digitalis was the only cardiac stimulant used; inhalations of steam and tr. benzoin for the first few days, with considerable relief.

Diagnosis: Influenza. Broncho-pneumonia. No complications.

Remarks: Discharged, recovered.

Case No. 15.—Medical Service.

C. G., male, age 28, single, porter, Italy, admitted February 20, 1919.

Past History: As far as could be obtained was negative.

Present Illness: Well until four days ago when he was suddenly seized with severe pain in right chest associated with headache and fever, no chill.

Physical Examination: General survey shows signs of consolidation, patchy in right lower lobe; rest of body normal. Urinalysis: Negative, save for faint trace of albumin. Temperature 104, pulse 120, respirations 40 per minute. Blood pressure: Systolic 112, diastolic 80. Sputum showed Pneumococcus type IV. The CO combining power of blood 44.8. Blood culture positive for Pneumococcus type IV, on February 21, 1919.

Clinical Course in Hospital: Patient remained in hospital twenty-one days. The pneumonia showed signs of resolution on the fourth day, but the following day his temperature again began to rise, reaching 10 on February 26th, with a similar rise in respiration. He showed signs of extension in right chest, with complicating pleurisy with effusions. Five days later patient's condition was much improved, temperature, pulse and respiration, normal.

Treatment: Fluid and farinaceous diet, fluids were pushed; use of regulation cardiac stimulation of digitalis, camphor in oil; codein to relieve pain; no thoracic paracentesis was done.

Diagnosis: Broncho-pneumonia, with complication of pleurisy with effusion.

Remarks: Discharged, recovered.

Case No. 17.—Medical Service.

F. W., female, age 24, single, clerk, U. S., admitted June 8, 1919.

Past History: Negative.

Present Illness: Well until twenty-four hours before admission; suddenly taken ill with usual signs of influenza, no chills, constipation and some headache.

Physical Examination: General survey shows nothing abnormal; pulse rapid; heart negative; lungs—respiration increased in rate, few scattered moist rales over both lungs, more marked in left; abdomen, negative; extremities, negative. Urinalysis: Negative. Temperature 103.8, pulse 120, respirations 35 per minute. Leucocytes 5,700, polys. 50%, Hb. 90%. Sputum showed Pneumococcus type IV. Blood culture positive for Pneumococcus type IV, on January 14, 1919.

Clinical Course in Hospital: Patient was in hospital twenty-two days. The temperature, pulse and respiration dropped at the end of thirty-six hours to normal; they began to show a gradual rise the next day and three days later the temperature was back to 104, pulse 120, and respiration 30. At this time there were definite signs of patchy consolidation over left lower base posteriorly; she developed an irritable stomach and became acutely ill. Temperature reached normal on twelfth day of stay in hospital and remained so. No complications developed.

Treatment: Ammonii chloride, ammonii iodide of each grs. $2\frac{1}{2}$ q. 3h., for the first three days; digitalis was given as routine for pneumonia; bicarbonate soda 5% sol. as drink; diet of general fluids.

Diagnosis: Influenza. Broncho-pneumonia type IV.

Remarks: Portal of entry, respiratory tract. Discharged, recovered.

Case No. 37.—Medical Service.

G. S., male, age 46, married, U. S., admitted November 4, 1921.

Past History: Negative.

Present Illness: Periods of intermittent fever for past seven weeks. Two days before admission, high temperature (105) and epigastric pain with chilliness. No gastro-intestinal symptoms other than the pain. No respiratory symptoms.

Physical Examination: General survey reveals poorly nourished man, who appears acutely ill; mouth presents several carious teeth, cervical glands enlarged slightly; heart not enlarged, no murmurs, but sounds were of poor quality; lungs show diminished ventilation in both, no rales; liver enlarged to level of umbilicus in midaxillary line; extremities, negative. Hb. 78%, erythrocytes 4,480,000, leucocytes 14,800, polys. 86%. X-ray report showed gastro-intestinal tract negative; chest, negative; frontal sinuses and both ethmoid regions show some clouding; teeth show several abscessed. Blood culture was positive for Pneumococcus type IV, on November 7, 1921, again on November 23, 1921.

Clinical Course in Hospital: Patient was in the hospital sixty-eight days. During this time the temperature varied from 99 to 104, sometimes a high A.M. temperature, other times it was high in the P.M.; the pulse averaged about 120 to 130; respirations 25 to 35. Blood chemistry was within normal range. He developed an irritable stomach soon after his admission and it was with difficulty that he retained his foodstuff. Sweating was a constant symptom. He gradually became weaker, pulse irregular, developed a general cyanosis, petechial spots in arms and legs, fine rales at both bases behind, and died January 10, 1922.

Treatment: Fluids were forced; carious teeth were extracted; sinuses were treated locally by nose and throat consultant. Salvarsan was used, intravenously, at regular intervals without any apparent effect.

Diagnosis: Pneumococcic bacteriemia, general.

Remarks: Patient died. Autopsy revealed: Chronic adhesive pericarditis (general); broncho-pneumonia; chronic adhesive pleuritis (left); arterio-sclerosis, general; arterio-sclerosis, kidneys; chronic passive congestion of liver. Microscopic examination was made of pericardium, lungs, liver, and kidneys.

Bacillus Typhosus Infections.

Case No. 19.—Medical Service.

J. S., male, age 27, married, real estate, U. S., admitted August 9, 1919.

Past History: Negative; he has never had typhoid fever and has never been inoculated against same.

Present Illness: Well until one week ago when, after eating soft-shell clams, he began to have general abdominal pain; the next day dizziness and general malaise, bloody stools, diarrhea, and fever; no chills, epistaxis, or respiratory symptoms.

Physical Examination: General survey shows well developed man; not suffering pain; no evidence of any cerebro-spinal irritation; tongue coated; no lymphatic enlargement; heart negative; lungs negative; abdomen—general abdominal tenderness, no rigidity or masses felt; liver and spleen not felt; extremities negative; no eruption on skin. Urinalysis: Negative. Temperature 104, pulse 120, respirations 30 per minute. Leucocytes 7,400, polys. 71%. Stool positive on August 11, 1919. Widal positive on August 15, 1919. Blood culture positive for typhoid fever, on August 11, 1919.

Clinical Course in Hospital: Patient remained in hospital sixty-one days. Temperature for first eight days remained high, between 102-104, after this there was a morning remission to 100, reaching normal on the twenty-seventh day, at which time his hemoglobin was 70%, erythrocytes 5,192,000. There were no complications during the entire stay in the hospital.

Treatment: High caloric, 3000 calories, soft diet; no cardiac stimulation was needed; daily enema was given; sponge baths while temperature was high.

Diagnosis: Typhoid fever.

Remarks: Discharged, recovered.

Case No. 26.—Medical Service.

K. R., female, age 26, married, admitted September 8, 1920.

Past History: Always of nervous temperament.

Present Illness: Began nine days ago with pain in chest and abdomen; severe frontal headache; epistaxis on two occasions; loose stools, two or three daily and of foul odor; appetite poor.

Physical Examination: General survey shows well nourished woman acutely ill; face flushed; oral cavity negative save for coated tongue; lungs are negative; heart not hypertrophied, slight systolic murmur at the apex; abdomen distended somewhat, no masses or

tenderness; liver and spleen apparently normal. Temperature 103, pulse 120, respiration 28. Urinalysis: Trace of albumin, few hyaline casts. Leucocytes 10,000, polys. 72%. Widal, positive for typhoid. Stools, positive for typhoid. Blood culture positive for Bacillus typhosus, on September 22, 1920.

Clinical Course in Hospital: Patient remained in hospital forty-one days, during which time the temperature averaged between 100 and 103 for fifteen days when it rather suddenly dropped to normal; the pulse averaged around 110 until the drop in temperature when it likewise slowed down to 90 per minute; no complications.

Treatment: Typhoid precautions; high caloric soft diet, 3000 calories daily; ice cap to head and codein for restlessness.

Diagnosis: Typhoid fever.

Remarks: Patient made an uneventful recovery. Discharged, recovered.

Case No. 28.—Medical Service.

C. R., male, age 29, married, manager of service station, U. S., admitted September 22, 1920.

Past History: Generally in good health.

Present Illness: For past six days complained of fever and abdominal distention, associated with slight cough; some headache; poor appetite; diarrhea, four to five loose stools daily, before that he was constipated.

Physical Examination: General survey shows head, negative save for infected throat and dry nasal mucosa; no enlarged lymphatic glands; no rose spots; lungs are negative; heart is hypertrophied slightly, the P_2 is quicker than the A_2, no murmurs. Abdomen reveals general distention, no masses or areas of tenderness, no rigidity; spleen palpable at the free costal border; liver not enlarged. Extremities and knee reflexes normal. Tempreature 104.6, pulse 90, respiration 30. Urinalysis: Negative except for trace of albumin. Lepcocytes 4,200, polys. 58%. Widal, positive for typhoid. Stools positive for typhoid. Blood culture positive for Bacillus typhosus, on September 24, 1920.

Clinical Course in Hospital: Patient remained in the hospital fifty-seven days. The temperature curve showed a continued tem-

perature between 102 and 104 for thirty days, when it began to drop by lysis reaching normal on the thirty-eighth day in the hospital. There were no subsequent elevations. The pulse never reached higher than a rate of 110 per minute. The patient was delerious at times requiring sodii bromide and morphine sulphate. Tr. digitalis was administered during the last week of high temperature; no complications save for weakened myocardium.

Treatment: High caloric soft diet.

Remarks: Discharged, recovered.

Case No. 29.—Medical Service.

P. A., male, age 19, single, laborer, Cuba, admitted September 2, 1920.

Past History: Negative.

Present Illness: Patient arrived from Cuba ten days ago and does not speak English. As far as could be learned, he had not been well on the boat but was seriously ill only two days before admission; he complained of general malaise, fever and pains in chest.

Physical Examination: General survey reveals man acutely ill; tongue heavily coated, breath foul; few scattered moist rales in posterior chest; heart shows systolic murmur at apex with slight hypertrophy; abdomen distended and slightly tender throughout; liver and spleen not felt; extremities negative; no signs of cerbrospinal irritation; no oedema; temperature 103, pulse 110, respiration 25. Urinalysis: Negative. Blood negative for plasmodium malarial. Leucocytes 36,000, polys. 89%. Wassermann: Three plus. Widal reaction: Positive for typhoid. Stools positive for typhoid on several occasions, still positive when he left the hospital. Sputum negative for T.B., many Streptococci and Pneumococci type IV organisms. Blood culture was positive for Bacillus typhosus, on September 25, 1920.

Clinical Course in Hospital: Patient remained in the hospital for sixty-four days. His temperature fell to normal after seven days in the hospital. There were no complications.

Treatment: High caloric soft diet during the course of the disease.

Diagnosis: Typhoid fever. Typhoid carrier.

Remarks: This patient was evidently ill for some time before coming to the hospital, and from the blood count together with temperature curve he had a central pneumonia but gave no clinical physical signs. Discharged from hospital as a typhoid carrier. Discharged, improved.

Case No. 35.—Medical Service.

D. L., male, age 16, single, U. S., admitted September 2, 1921.

Past History: Negative.

Present Illness: Complained of fever, headaches, and general malaise for two weeks before admission, these symptoms followed upon a visit to the country.

Physical Examination: Revealed many "rose spots" over chest and abdomen; abdomen was tympanetic throughout; lungs were negative, save for few sibilant rales; liver and spleen not palpable. Urinalysis: Negative. Leucocytes 5,200, polys. 56%. Stools were positive for typhoid. Wassermann: Negative. Widal: Positive. Blood culture positive for Bacillus typhosus, on September 24, 1921.

Clinical Course in Hospital: Patient was in the hospital forty-two days; he ran a temperature between 102 and 104 with a pulse of 110, respiration 30, for sixteen days when it gradually became normal by lysis on the thirtieth day in hospital. He had no complications and made an uneventful recovery.

Treatment: Usual high caloric diet; no medication administered; high calonic irrigations at times for abdominal distention; ice cap to head for headache.

Diagnosis: Typhoid fever.

Remarks: Discharged, recovered.

Bacillus Aerogenes Capsulatus Infections.

Case No. 30.—Surgical Service.

H. P., male, age 50, widower, U. S., admitted December 27, 1920.

Past History: Negative.

Present Illness: Well until eight days ago when he stuck his left forearm with a sewing needle; next day redness and swelling extended to axilla and he suffered considerable pain; some headache but no chills.

Physical Examination: General survey shows left forearm swollen, oedematous from shoulder down to hand; on posterior surface of forearm is an opening, the orginial wound from the sewing needle, some discharge. Heart, lungs, and abdomen, negative. Temperature 102, pulse 128, respirations 20 per minute. Blood culture showed anaerobic culture of Bacillus aerogenes capsulatus, on January 9, 1921.

Clinical Course in Hospital: Patient remained in the hospital sixty-nine days. On December 28, 1920, was given nitrous oxide gas anaesthesia and free incisions with drainage were done; cultures taken from infected area on January 4, 1921 showed Bacillus aerogenes capsulatus (Welch's gas bacillus). On January 4, 1921, for uncontrollable sepsis, amputation of left extremity just below insertion of deltoid muscle. Anaerobic cultures from separated deltoid muscle show Bacillus aerogenes capsulatus (B. Welchii). Specific Welch-Tetanus serum was given intravenously as follows: January 5, 1921, 180 c.c. of specific serum, January 6, 1921, 100 c.c. of specific serum; January 7, 1921, 100 c.c. of specific serum. Temperature 100, pulse 130 before serum; temperature 100, pulse 100 after serum. Blood examination on January 6, 1921 showed Hb. 35%, erythocytes 1,400,000 to cubic mm., leucocytes 37,000, polys. 95%. Two transfusions of citrated blood were given, the first on January 8th, the second January 15th. The blood count on January 19, 1921 showed Hb. 55%, erythocytes 2,800,000; on February 23, 1921, Hb. 70%, erythocytes 4,060,000. The patient's temperature at no time went above 103, the pulse was very unstable, fluctuating between 80 and 140. The heart showed only myocardial disturbances and he was discharged, improved, March 6, 1921.

Diagnosis: Cellulitis of left arm.

Remarks: Discharged, improved.

DISCUSSION.

The President: I will ask that Dr. George Norris of Philadelphia open the discussion on this paper.

Dr. Norris: I have been asked to discuss this paper on short notice. I do not feel competent to discuss it, except in a general way.

Of course, the class or cases to which he has alluded are those which show up more than anything else the importance of the medical profession. From

time to time drugs have been vaunted as a cure, and practically all of them have been sooner or later given up.

In regard to magnesium sulphate, I personally have had no experience. In Philadelphia, a number of cases of sepsis have resulted from child birth, and a drug called marsone apparently has been used with good results. In regard to the details, I am not well informed on them, and not enough cases have been treated which would warrant a definite conclusion. Whenever we hear favorable reports following the treatment of any definite or specific substance, I think we think perhaps it had nothing to do with the cure.

I think, just as we have learned in recent years, that bacteria will not always produce general peritonitis, as the peritoneum can take care of them, so there has been a feeling that if bacteria gets in the blood it does not necessarily mean septicaemia. There are a good many cases where general bacteriemia exists, which would give negative results on blood culture. There are classes of this kind of course, which makes us think the condition of bacteriemia is a good deal commoner than is generally supposed.

Dr. Babcock: I have been very interested in this paper, and in the first place I want to say a few words with reference to the tonsils as a focus for infection. In the last quite considerable number of years I believe in my office we have never yet seen a child or a young person in whom proper examination or history did not disclose an infected tonsil. I have come to look upon tonsilitis in children as an exceedingly serious matter.

With reference to the abscesses also, I regard them as exceedingly serious, as I have had some very interesting cases.

I can recall at least two men sent to me with a diagnosis made by very competent men, and two of them in a very renowned clinic in the west. Both of those men have apparently a typical description of anginapectoris. Both of those men had abscesses, and when their teeth were extracted and properly treated, both of those men recovered from the anginapectoris. With reference to the streptococcus it certainly does attack the valves. It also attacks the aortic valve.

I have seen a case of primary carditis which was due to streptococcus, but I am constantly running across cases, malignant cases, in which the history on examination showed that they had a previous injured valve. I advise removal of tonsils as a preventative measure, if possible. It is not a guarantee, of course, but it often is paramount with reference to the vaccine treatment. I have a man in my care, and he was given the most careful treatment, vaccines over two months, and everything seemed to be progressing, but after that he died from hemiplegia. I used sulphate of magnesia, and it does not do any good.

The viridans is an organism which gets in the blood stream so much so that if you want to get it—take your blood when the temperature is at the highest, otherwise you will miss it frequently—when the organism is finally circulating you will get your culture, if your laboratory worker understands. Therefore, when I use sulphate of magnesia, and the bacteria was apparently kept under control, I did not feel sure it was due to sulphate of magnesia. I thought the

patient's blood destroyed the organism, but the patient died, and every one has died, and I am skeptical about the possible recovery, although such cases are reported of viridans carditis.

I can cite a number of cases where patients had a lesion and did not know they had heart trouble until they got tonsilitis, and developed carditis. I think tonsils and teeth are portals of infection that we have got to watch, although portals of entry must exist elsewhere than those two.

ABSCESS OF THE LUNG AND THE METHOD OF PREVENTION IN NASO-PHARYNGEAL SURGERY.

By CHARLES H. RICHARDSON, M.D.,

WASHINGTON, D. C.

I feel somewhat as though I am going a little beyond the sphere of activity of this Society in bringing before you the paper I am doing, but this is a burning question with us, and there are some facts in relation with it that I think are of vital importance in a clinical way.

As you probably know, I have not been an extreme tonsillectomist. I have always tried to find out why a tonsil should be removed. I have always wanted it condemned and adjudged properly before I would be the executioner.

I have over and over again questioned my internist, who sent me the cases, why he did it, that my judgment did not coincide with his, and I have often been subject to the loss of clinical material from certain practitioners, because I have not carried out their judgment, so I want you to understand that I am not an extremist.

I want it also understood that there are certain features of this work that I believe in most thoroughly, and when I do it I do not do it except in my way. I would rather lose the patronage of a half dozen physicians than subject a patient to dangers which I know we can avoid. I will not let the laiety suggest to me the way I will do my work. I do it my way, and my way only.

I have been doing local operative work long before local operative work was suggested, and I gave it up. I did a local this year, only a few months ago, but I feel the only way to do these operations safely is with proper, thorough protection, with a skilled anesthetic ist, to do the work as well as you do yourself.

General anesthesia in this work has been subject to criticism, because any Tom, Dick and Harry, who knows nothing about it, has been employed to give an anesthetic to a case of this kind, which requires the most skilled and best prepared anesthetizer you can find

to do it. Under such circumstances there is no question that general anesthesia is safer, saner and gives the best results.

Over in the Laryngological yesterday men talked about giving anesthesia, but not destroying the cough reflex.

In every case of local anesthesia that you necessarily destroy the cough reflex, the safeguard of the patient, and moreover, they stated in order not to destroy this they would rather their patient suffer pain. Good heavens, gentlemen, did you ever hear of a man of any heart doing a safe and sound operation when he knows his patient is squirming and prays to be let alone! "For heaven's sake, do not touch the other one," I have heard them cry in an adjoining room when I was operating in the other room.

I have over and over again asked my assistants, when I was operating, what was crying out and screaming. "That is one of the safe and sane and painless, bloodless, local, tonsillectomist."

Remember, gentlemen, when you condemn this work or criticise it, remember that you have got an area that is very difficult to operate on, that it requires manipulative skill, surgical dexterity, and surgical intuition. A man must know thoroughly his anatomy; he must thoroughly know what he is going into, and how to cope with it. I find that too many men criticise my methods and results. If they only knew more they would not criticise so severely. The man who has done a thousand tonsillectionies, has just begun to learn. It takes a man who has done five thousand before he knows how to do it and control every factor which might be resultant to the future welfare of his patient. It is one of the most dangerous regions in the human body to operate in.

I read a paper over in the Laryngological on what we call a well performed tonsillectomy. I hope you will read it.

There are conditions which arise which no human being can control in these operations of a local character. You must remember that the lymphatic tissue is a tissue that resents its destruction in early life, that it will reproduce itself not only in the immediate area which is operated upon, but in the whole area around the wall and pharynx in the anterior pillar, the posterior pillar, in the latteral of the tonsil, and that tissue will reproduce itself.

I have seen cases five and ten years after, not mine own, but

in the work of other men, who are the best workers in this country, that were absolutely clean, ten years afterwards, which show large recurrent tissue growth. Now, gentlemen, there was a case reported by a doctor, several cases reported yesterday, of the same kind. That is not granulation of the tissue, which you overlook, but by study it is proved not to be tonsular tissue, but lymphatic tissue, with lymphatic lodes in it. Where did it come from? Where does it come from? It grows over and works up the latteral wall of the pharynx. They lay dormant and swell out and show activity. In the same way they come up over the posterior pillar. I have seen cases in which the posterior pillar, in most beautiful fauces, not my own, excellent operators, men that we all vouch for in knowledge and surgical skill; I have seen posterior pillars very large, and laying so far forward, unless you throw the pillar backward you would think it was a tonsil, yet when you threw it back there was an absolute, perfect looking fauca as you ever saw. All men who do this work know it and see it.

May I read my paper?

If anyone is going to discuss this paper I want you to pay attention to some few points, that unless you do, you may criticise in the wrong way.

There is no question that abscess of the lung has been too frequent a complication of operative intervention in the upper air tracts. This is partly due to other important facts, namely, that in most of the operative procedures in the upper air tracts we have to do with infected material, the escapage of which we are not always able to control, and whose escapage is facilitated into the area to be secondarily infected by a most perfectly arranged open tubular system.

There is only one God, and that is the superior stricture nerve, which controls the sensation in the lower pharynx, and the subcardial portion of the larynx. If that is gone, nothing you do will prevent the fluid from getting into the lungs.

The process of infection in the large majority of cases is hardly open to argument. Infection takes place in the larger percentage of cases to the direct inspiration of infective organisms, contained in fluids, pus, small particles of cheesy material, small masses of tissues

or other debris. There is probably a small percentage of cases occurring after tonsillar operations wherein the infection takes place through the lymphatic or vascular system. There is no doubt that an operative seance done under the unconscious state, without a well conducted technique, is more productive of this occurring than is the same operative procedure done under the conscious state.

We are, nevertheless, learning through reports of cases of operations done in the conscious state under local anesthesia are not immune from pulmonary infection as well as other forms of infection, which do not transpire under the unconscious state. I wish you to catch that. All of us know it. They are published facts. Now, the facts are coming out. A lot of us know these things.

Six years ago Matthews of Rochester told me he had six serious almost fatal abscesses in the neck following local anesthesia. Dr. George B. Wood, yesterday, in the one little town, the village on the Delaware, called Philadelphia, had had between twenty and thirty cases of serious local infection following local anesthesia in tonsillectomy, local anesthesia, three of which died, and all of which lost from three weeks to two months as a result of this local infection from local anesthesia.

Gentlemen, this is only a small tale from one town. That does not include over fifty instantaneous deaths from local anesthesia, sitting in a chair, three minutes afterwards down on the floor dead, with no possibility of ever restoring. Why? Because you injected one or two drams of a solution which is only begun to be absorbed when your patient is dead. Before you can do anything there is still more going in. There is only a little absorbed when the patient is dead. You can save them if you get in danger. There is another point. This is not in the paper. I am just talking. I think you ought to know the facts.

Dr. Moore of Philadelphia, one of Dr. Jackson's assistants, told me he was going to conduct an investigation of the number of pulmonary abscesses following a local and general anesthesia. I know some of you will smile when I say pulmonary abscess. His paper is published. It occurred in the last number of the Journal of the American Medical Association. It is a most illuminating thing. He dug up thirty-nine abscesses of a lung from local anes-

thesia, and that is nothing. I could almost say, if the man had been honest, he probably could have gotten one hundred or more, or probably two hundred.

Here in Washington there have been four who have never seen the light of day, and none of you know about them, and the men who had them will not let you know. That is against 121 under general anesthesia. These 121 under general anesthesia date back to the days before proper corrective methods were adopted. So you see the local anesthesia is coming up pretty close to the general anesthesia.

There is one interesting thing in this report. Most of the cases of pulmonary abscess occurred in New England states, where they are under the influence of Bostonian atmosphere.

Why in heaven's name they take a man and sit him up in a chair and slap an ether cone and anesthetize him, and take out his tonsils, or even do such operation with a man in a sitting position—I cannot understand it. It is beyond my comprehension they do not kill them all.

There is a case of pulmonary abscess which occurred in New England, under the Boston atmosphere. That is one thing they do, tell the truth and shun the devil. I go to Boston every summer.

Dr. Babcock: How about Chicago?

Dr. Richardson: Chicago? I know it too well.

It would lead us into too long a discussion to give the reasons one may conscientiously have for preferring general to local anesthesia in faucial operations. Sufficient is it to state that the operation can be done safer, better and with less risk to the patient under general anesthesia. General anesthesia (ether) in the hands of a skilled anesthetist should be absolutely safe to life. Local anesthesia as applied to producing anesthesia in the fauces has already demonstrated that it is a dangerous agent to the life of the patient.

It is not my purpose to present the manifold reasons other than those generally outlined above why general anesthesia is preferable to local anesthesia in faucial operations; as the main object of this paper is to demonstrate how, through carefully conducted technique, under general anesthesia, the occurrence of pulmonary abscess in faucial surgery can be eliminated. I mean eliminated.

The technique which I have adopted and which seems to have eliminated this type of surgical risk in faucial surgery is as follows:

First. A careful pre-operative preparation of the patient.

Second. A medical anestheticist, skilled in the use of the Beck or similar type of anesthetic or suction apparatus.

Third. The placing of the patient in the moderate trendelberg position during the operation. Head and shoulders well dependant.

Surgical cleansing of the place and removing all escapage in the tonsil, and giving them proper doses of soda and lactose, and all those things is important.

I do not let my anesthetist drown my patient. The pharanygeal reflexes are slightly benumbed, but none destroyed. The larynx is under control. They cough, and sometimes they spit, before they are removed from the table, and I often get it in the face.

Fourth. The anesthetist skilled in following the knife and desector, or other pressure about the fauces so as to gather up through suction, pus, sees the material or liquid escape from the tonsils.

Fifth. The most gentle manipulation in applying the pressure and swabbing.

Sixth. The patient is returned to the bed resting on left side, and should be placed in bed face downward until thoroughly reacted.

Through the careful carrying out of the above technique I have had a perfect relief from this unpleasant complication in faucial operations during the past years, although I have performed more than my usual ratio of adult operations.

My secretary went over my operative book, and found there were more than double the cases over the last four years, of adult operations, more than double than I had done in any four years preceding period.

In the nasal and naso-pharyngeal operation, which from its major character we are accustomed to resort to general rather than local anesthesia, it is not surprising that we occasionally have a lung abscess as a complication. Until the occurrence of this complication, following some of these operations became a subject of comment, the operative technique was not universally employed. To safeguard our patients should be one of the cardinal principles in

surgery. To do the operation well, skilfully and quickly is not difficult. It is only through the employment of great care that we can prevent leakage into the lower air tracts.

In operating upon the antrum, frontal, sphenoid and post-ethmoid cells, filled as they are with infective material, it is imperative to block all possible means of the escapage of pus, granulations or other debris into the lower air tracts through proper placed post-nasal tampons.

Remember in these cases that we have pus and broken down materia, which goes into the nasal chamber, and it is surprising that more does not leak down.

The nasal chambers should be well cleaned before the post-tampons are removed. The post-tampons should not be removed until the patient has reacted sufficiently to give evidence of self protection.

In the radical operation on the antrum through the canine fossa route, it is important to place a large tampon in the buccall cavity in order to prevent leakage from this area on into the lower air tracts. The patient should be protected by posture after returning from the operating room until he has completely reacted.

Pulmonary abscess is such a typical and thoroughly characteristic condition in its etiology and symptomology that one should not mistake it for any other disturbance.

I am going to tell a story. A nurse in New England, in Boston, went to Boston with pulmonary abscess. The doctor denied it, said there was no possibility. She went to another doctor who said it could not be. She was a nurse in a surgeon's office, and when she told him he said she must have an abscess. They x-rayed here and she did.

I read a paper at the A. M. A.; she heard it and asked me if she had tuberculosis or pulmonary abscess, and she had the most beautiful characteristic picture of pulmonary abscess. No one could given any better.

That such mistakes are, however, frequently occurring is the only excuse I can offer in closing my paper with the presentation of the symptomatology of this disease. In most cases you have an operative procedure of a more or less infective area in direct com-

munication with the lower air tract. The invasion may occur immediately or be delayed three or four days after the operation has been done. If the invasion is apparently made the patient does not do well in the interval between the operation and the first signs of invasion. The actual invasion is usually manifested by a chill or chilly sensation, followed by a rapid rise in the temperature, which immediately assumes a septic character. Pain in some area of lung is early present. Cough, paroxysmal in character, is a very early symptom. Odor of the breath, first noticed by the patient, later by attendant, is quite a characteristic symptom. Profuse heavy pus expectoration, which may become rusty as the case progresses. Hemorrhage may occur. Profuse sweating at night. Temperature, pulse and respiration run in usual ratio. The earliest physical signs are those of an infiltrated area, frequently unrecognizable in the early stage, becoming more clearly defined on successive days. The radiograph gives an absolute picture which cannot be misinterpreted. The most important element in treatment is early diagnosis. The early production of artificial pneumo-thorax is productive of the best results. If the abscess is discovered too late, illy situated for pneumo-thorax, or of such nature that rupture may take place under its application, then surgical methods must be employed in treating this unfortunate complication.

May I take one word more?

THE PRESIDENT: Yes, you may continue.

DR. RICHARDSON: I know I am imposing, but I want to say one thing here about pneumo-thorax. You know if you wait until the very last stage, then the walls become dense in character, but if you use it early in your case you almost always get splendid results. I do not know anything more beautiful than to see temperature drop and signs improve, etc. Physically, I find these cases are easier after you learn the trick once or twice. I had a case two years. In the first place there as a pain in the right side, at the second or third rib. This was the second day, after a chill and temperature. I examined, and there was just a little infiltration. I examined that case three days afterwards, and it was in the lower portion of the base. The case was x-rayed, and there was an area

here (indicating) where it started. The bronchi could not hold it, and here was the abscess (indicating).

There are so many things on this subject that I could talk about a week; there is another important thing to prove, this is an inspiration or call it exporation, in that we find that it is almost on the right side, and on the upper or lower lobe, and the ratio is almost identically the same with the ratio in the lung.

DISCUSSION.

THE PRESIDENT: This very instructive paper is now before you for discussion.

DR. MINOR: The doctor used a good deal of time in the discussion of local and general anesthesia. This is hardly a thing to be discussed here.

As regards the question of abscess: The early diagnosis is overlooked. I agree with the doctor that if it is made early and properly done a pneumothorax is an ideal operation. It will not work every time, and secondarily he states an x-ray is always positive. By no means. When it is, it is absolutely satisfactory, but I can show you many cases where you cannot positively make a diagnosis from an x-ray picture. You can make it from symptoms. The trouble is that the nose and throat man does not follow his case up closely after the operation, and generally the first symptom that attracts you is the discharge of pus. That comes if pus has collected some time later.

I do not see them in their earliest stage. Our local anesthetists have been successful in their work. I have had as many come to me as four in two months, terrible cases, beyond any question of a cure. It is not an easy thing, and we ought to look upon tonsillectomy as a major operation. An operation which can produce a disease which is fatal at times is distinctly a major operation. With care on the part of the operator at the beginning, and keeping the patient in bed at least a week afterwards—all of this should be done. Keep your patient quiet in bed. If the head is kept down, I will not worry about local or general anesthesia. The head should be kept down, and Dr. Richardson is responsible for that position, and for that we owe to Dr. Richardson a great debt.

All I would say is that I think the statements in many of the other things, which are debatable, are too positive. We must remember that an intense conviction is apt to carry us beyond what is a fact. His positive statement on x-ray is wrong. His positive statement as regards pneumo-thorax also is wrong. Those positive statements in medicine are dangerous, but if we follow up the many points the throat man and the general practitioner offer, it will be helpful.

DR. BRANNAN: I wish Dr. Richardson would come to New York and not to New England.

I started out in New England a good many years ago. They had the highest respect for ether. This was in the Massachusetts General Hospital, and

they did not think the members of the staff could administer ether without some one watching over us.

I can still remember the formula, pull out the tongue, head forward, vasin, a little more air, so on and so on.

I wish he could have come to New York twenty years ago. I have not done much of this tonsillectomy; I have not done it for thirty years. About twenty years ago I visited Bellevue Hospital, in the operating department. We had a request for some supplies. I passed down the waiting room, and saw a number of children and their mothers sitting at the door of a closed room, and inside that room I heard shrieks of terror and pain, and took the liberty of opening the door, and saw the child sitting on the lap of a nurse, in the old fashioned way, grasping the knees and arms, and that child was having tonsillectomy, first one side and the other done, without any anesthesia whatever. They did not know so much about local anesthesia in that part of the body.

I said, "Why don't you give these cases ether?" She said they had no wards for them.

We stopped that work then and there, and in two or three weeks we found a comfortable ward in which the children were kept from twelve to twenty-four hours before the operation, and from twenty-four to thirty-six hours after the operation.

At that time in the special hospital at Manhattan, New York, the practice was the same. If any man is here who knows to the contrary, I wish he would say so.

I never tried to introduce any reform, without talking to men doing the same work in private hospitals. I talked to Bacon and others, and I found in private hospitals they had come to the same conclusion. All that was needed was for somebody from the outside to see what was going on.

This reduced the number of operations. At the same time we were under the impression that there were still more in the schools who wanted to have this done, and thousands from the schools desired to have it done. The Board of Health did the operations and give it up. We do now from three to four to five thousand operations in the course of the year, and a waiting list at Bellevue of three months.

I never heard of pulmonary abscess, but I knew that children under this improved method of treatment do go home and suffer four or five pounds loss in weight, have constipation, digestive disturbance and so on.

I call this a major operation. Dr. Minor spoke of that. It was difficult for me to make the throat man consider it a major operation.

Dr. Babcock: I have no quarrel with Dr. Richardson as to the kind of anesthesia. I think it is a question of the operator rather than the particular form of anesthesia. There are few internists I think, with limited experience as myself, who have been responsible for relatively so many tonsillectionies, and from my experience this is—if I recommend a tonsillectionomy it has got to be done by somebody whose technique I know about, and if a patient says he is going to a family doctor, "I will have no responsibility with reference to it."

There are too many family doctors and general surgeons doing this operation, in my opinion.

It is the technique of the particular operator. I have had patients operated from three years to sixty years, under both forms of anesthesia, and the man whom I select for that work have never yet had a pulmonary abscess or an abscess. It is the operator and his technique, and not the particular form of anesthesia, in my opinion.

Dr. Wilson: I do not think enough stress was laid on the embolic side. I have had nine cases of lung abscess, eight of which were post-operative; three followed tonsillectomy, and one ethmoiditis. We will consider the question of those cases following tonsillectomy. In all of those three cases the first symptoms arose after the first week of the operation. In three cases it was after the patient returned home. The patient remained at the hospital three nights following the operation. One of them is a great deal of interest. He was operated on in Washington, returned home ten days afterwards, and ten days after returning home he had a chill, a cough, and from that time on, and he came up six months afterwards he was pretty well knocked out.

He recovered from the lung abscess, but died from brain abscess. I think a great number of those cases are essentially embolic. That has been reported from Johns Hopkins. Going into an analysis of cases, they found a large number of the cases were embolic.

The President: We will now hear from Dr. Elliott.

Dr. Elliott: I just want to mention two points, Mr. Chairman. The first series of cases that I saw were practically all after general anesthesia. Within the last year I have seen two follow local anesthesia. When we remember there are 100 cases under general for one under local, we will find in pulmonary abscess a large proportion follow general anesthesia, and if we take out the proportion of lung abscess following general, as compared with local anesthesia, and consider the large number of operations done under local and general anesthesia, we will find more under local than under general.

In regard to the time element, I have seen a patient have a chill immediately after coming out of an anesthetic, and taken into the ward with a temperature of 104. Most of the cases arise in three or four days, but I have seen cases where symptoms of lung abscess, no cough, or anything develop until thirty days after the operation.

The President: Dr. Richardson, will you close the discussion?

Dr. Richardson: Dr. Minor considered my statement a little positive, but I want to read a line right here. "The early production of artificial pneumothorax is productive of the best results."

Dr. Minor: After that he said it was an absolute cure.

Dr. Richardson: No, I did not. I said: "If the abscess is discovered too late, illy situated for pneumo-thorax, or of such nature that rupture may take place under its application, then surgical methods must be employed in treating this unfortunate complication."

That is all the treatment. Nor did I in any way say that general anes-

thesia was to be preferred to local anesthesia. Nowhere did I say that in my paper.

If I have been a little positive in my statements, it is borne out probably by judgment that comes to a surgeon after long experience in operating work. It is a something indefinable, which you cannot understand, but it is there.

There are certain cases which we would well operate under local anesthesia, and there are some that we never would. There are certain cases we would not give general, and prefer to do it under local anesthesia.

Why are these abscesses in the tonsils and neck so frequent after local anesthesia? Simply because you drive a syringe through an infected organ, and carry the infective micro-organism with the point of your syringe, and inject it into the tissues. That is frequently carried into the substance, all through this muscle, and when you carry it in there you carry it in there, that which will surely be followed by an infection. It is not so much the technique—it is the technique in a certain way, but it is one of those things you cannot account for. A man will carry his needle a little too far away sometimes and produce them. You might say certain men do not have certain conditions. When you get to a close personal contact you find we do have things you do not suppose they have.

As to Dr. Brannan's remarks, I think they are splendid. There is no question that this is a major operation, and most men that do it consider it a major operation. I have heard the best men from all over the country state that they would rather do a mastoid any day than to do a tonsillectionomy.

DIAPHRAGM IRREGULARITIES
(Preliminary Contribution)

By RALPH C. MATSON, M.D.

PORTLAND, OREGON.

During the course of the examination of registrants prior to entrance into military service at Camp Lewis, considerable numbers with lung abnormalities, including what was considered obsolete or healed turberculosis, were accepted for military service by the Tuberculosis Board.

Careful records, including physical examination and roentgen ray findings, were made of more than 40,000 out of the 174,212 soldiers examined by the board on entrance into service, during service and prior to separation from service. These records included all cases in which tuberculosis was suspected either from the general condition of the registrant, his past or present history or from the results of the physical or roentgenological examination.

In order to check up our judgment in accepting registrants with lung abnormalities for service, especially those who had what we thought was healed tuberculosis of no military importance, we decided, among other methods of control, to examine every soldier who had been in the base hospital on account of measles, whooping-cough, influenza, pneumonia, broncho-pneumonia, and other respiratory infections. These men were examined at once after discharge from the base hospital, again at the end of a month, and we endeavored to make a third examination approximately two months later. Of course, not all soldiers were available for the three examinations. However, we followed 3,754 cases of the above character who had been in the base hospital during their stay at Camp Lewis, and later during demobilization we were in a position to compare their examination on entrance into service and examination made after discharge to duty from the base hospital, with the physical examination made prior to separation from service. In addition to the above we were able to compare the entrance and demobilization

examination of an additional 6,064 cases, who had not been in the base hospital but who presented lung abnormalities on entrance into military service. These examinations revealed diaphragm irregularities under the fluoroscope and on the plate in four per cent of all cases studied.

Heretofore, irregularities of the diaphragm having a tented appearance have been thought to be due to pleuro-diaphragmatic adhesions. However, in checking up examinations made at various times on the same cases, some observations were made which were inconsistent with the assumption that tenting of the diaghragm necessarily meant pleuro-diaphragmatic adhesions.

The following case is a typical example of observations which prompted this clinical study:

J. H. B., aged 22 years, private in the infantry, was examined June 28, 1918, upon his arrival at Camp Lewis for military service. The fluoroscopic examinations showed the diaphragm regularly curved, phrenicocostal angles clear, good excursion and no delay. On November 26, 1918, he received his first examination after discharge from the base hospital where he had been confined on account of an influenza-bronchopneumonia of the lower lobe of the right lung. The fluoroscopic examination at this time showed "delayed diaphragm excursion on the right side with tenting of the middle third at the termination of the hilidiaphragm superfices." This was attributed to pleruodiaphragmatic adhesions, the result of his previous broncho-pneumonia. He was examined again after one month, showing practically the same findings. The third examination was made three months later, and while the report stated there was some exaggeration of the hilus and hilidiaphragm superfices, no remark was made regarding tenting of the diaphragm.

As similar findings had been noted in other cases, it appeared that either the fluoroscopist was overlooking abnormalities or failing to dictate them to the stenographer who took the dictation in the dark room or the stenographer was careless in taking or transcribing his notes. Therefore, to determine, if possible, where the fault lay the case referred to (J. H. B.) and other cases wherein errors were thought to have occurred were called up for examination, as a result of which the correctness of the findings of the last previous fluoro-

scopic examination was confirmed. In the case of J. H. B., no tenting on the diaphragm was found upon re-examination. Case records were gone over and other cases of the same character were studied anew and it was found that many individuals presenting tenting of the diaphragm on one examination showed no tenting at subsequent examinations made after an interval of several months.

Accordingly, the inference that pleurodiaphragmatic adhesions were responsible for all these irregularities of the diaphragm characterized by tenting was no longer consistent, because in many cases the irregularity disappeared, which would not have been the case where pleurodiaphragmatic adhesions the cause.

After some study the following theory suggested itself as a logical explanation for the occurrence of the type of diaphragm irregularity appearing as a tenting at the termination of the hilidiaphragm superfices which we had seen disappear during the course of our fluoroscopic examinations covering a period of several months.

The lower bronchial branches may be likened to rubber tubes stretching and contracting as the lung expands and contracts in response to a downward pull and rise of the diaphragm. During the course of acute and chronic respiratory infections, wherein infiltrative and proligerative changes occur in the bronchi and peribronchial tissues, the normal elasticity of the bronchi is diminished. If these changes are slight and do not extend to the superfices, the downward pull of the diaphragm may be only delayed or retarded (possible explanation of William's sign), or if the process has extended to the finer bronchi and is of a diffuse nature, the diaphragm frequently assumes a wavelike irregularity. However, in cases in which the bronchial and peribronchial changes are more extensive and involve the respiratory bronchioles situated close to the basal pleura the downward pull of the diaphragm meets with response on the part of the lung base everywhere except over the area served by the branches involved, frequently the termination of one of the lower lobe bronchial branches. Over this area the diaphragm fails to pull the lung downward or at least not to the extent it does elsewhere. Accordingly that area of the lung base to which the bronchus leads is more or less fixed by the rigid bronchus, which acts as a buried suture extending from the hilus to the basal

pleura. The negative intrathoracic pressure holds the diaghragm firmly to the lung. Consequently, during inspiration traction of the bronchus resisting diaphragmatic pull produces a dimple on the surface of the base of the lung. The diaphragm, being unable to pull the lung down at this point, must necessarily mold itself to the irregular contour of the base of the lung and is, therefore, sucked into the dimple, thus presenting a tenting which we will designate pseudo-adhesion.

The idea that the intrathoracic negative pressure is sufficient to suck the diphragm into a lung dimple is supported by the Sahli's explanation of Litten's phenomenon, which according to Sahli is due to suction exerted upon the intercostal spaces as the diaphragm begins to peel off the thoracic wall in its descent. The fact that the soft parts of the thoracic wall are sucked in during inspiration by the "peeling off" of the diaphragm, which tends to create a vacuum, leads us to believe that the intrathoracic negative pressure is sufficient to hold the diaphragm firmly to the lung and conform it to the contour of the lung base with which it comes in contact. These irregularities due to pseudo-adhesions appear to be more marked in the asthenic types in which the diaphragm has undergone atrophy and suffered loss of tonus as has the other musculature.

Anatomical support for the theory above advanced was found in the following case coming to autopsy at General Hospital 21, Denver, Colorado, during my service there.

H. J. N., aged 29 years, private in the infantry, had pneumonia in November, 1917 (side not stated), and made an apparent recovery. He again entered a hospital with "pneumonia" June 4, 1918, and was continuously hospitalized thereafter, being transferred to General Hospital 21 April 21, 1919, with a diagnosis of chronic active fibro-caseous tuberculosis of the upper lobe of the left lung.

For the purpose of this paper it will be sufficient to quote only the roentgen findings referring to the diphragm in this case. The roentgenological examination of May 1, 1919, states:

"The diaphragm on the left side presents marked tenting in the midclavicular line due to pleurodiaphragmatic adhesions. Subsequent examinations made on September 20 and December 20, 1919, showed

no change in the diaphragm irregularity. Exitus took place February 2, 1921."

The only part of the autopsy report which concerns the subject under consideration is the pathological findings of the left lung and the diaphragm:

"Autopsy revealed the left lung adherent at the apex to the dome of the thorax and to the pericardium above. *The base of the lung was smooth and glistening and no adhesions to the diaphragm were present.* The cut section of the lung showed several cavities in the upper lobe with disseminated tubercles in the lower lobe. The lower lobe bronchial mucosa was studded with tubercle to within one-half inch of the pleura. The lower lobe bronchi showed marked connective tissue proliferation with peribronchial infiltration extending to the diaphragm."

Diaphragm irregularities due to pseudo-pleuro-diaphragmatic adhesions appear in general, from the limited material studied, to be sharply defined, angulated tentings occurring at the termination of the ramifications of the lower lobe bronchi, the tenting frequently being an apparent continuation of the bronchus. On the other hand those irregularities which are diffuse, not so clear cut, nor so angulated and so closely related to the bronchial ramifications and not following the anatomical course of the bronchi appear to be due to pleurodiaphragmatic adhesions.

The case outlined above is the only one having an irregularity thought to be due to pseudo-adhesions which we have had an opportunity to study at autopsy. However, the utilization of artificial pneumothorax for differential diagnostic purposes has furnished information of a nature confirming the theory advanced. During the past six months we have been able to study eight patients presenting diaphragm irregularities having the characteristics we have associated with pseudo-adhesions. The procedure carried out was the introduction of sterile air into the pleural cavity to release the suction or at least to reduce the intrathoracic negative pressure. All cases were fluoroscoped and stereoscopically plated beforehand, at once after and every few days for two weeks or more. In some cases the air was introduced while the patient was studied under the fluoroscope.

Of the eight cases studied we believe six are pseudo-adhesion cases. The following case is typical of the six:

L. B., aged 17 years, was first seen February 12, 1918. Her chief complaint was cough and expectoration dating from an attack of whooping-cough followed by pneumonia four years previous. She had never complained of pleurisy. The physical examination was negative. The roentgen plate showed slight exaggeration of the hilus shadows with increased density of the lower lobe bronchi, more marked on the left side. There was a tenting of the diaphragm at the termination of the hilus-diaphragm superfices on both sides. The patient was seen again August 16, 1920. The physical examination was negative. The roentgen examination revealed no changes from the examination two and a half years previous. According to my new conception regarding diaphragm irregularities this case had an irregularity presenting characteristics of the pseudo-adhesion type. For differential diagnostic purposes the introduction of a small quantity of air into the left pleural cavity was decided upon. Accordingly a pneumothorax needle was introduced and the manometer recorded a negative pressure $1\frac{1}{2}$ to $3\frac{1}{2}$. Sterile air was introduced without pain under fluoroscopic observation. As the air flowed in the irregularity of the diaphragm gradually disappeared, being entirely absent after 250 c.c. of air had bene introduced, at which time the intrathoracic pressure measured $1\frac{1}{2}$ to $2\frac{1}{2}$. The plate made immediately afterward revealed a regularly curved diaphragm and the tenting had completely disappeared, yet the quantity of air was so small that it was only apparent at the dome of the thorax; nevertheless it was sufficient to release the suction between the diaphragm and the lung. Fluoroscopic examinations were made at intervals of every three days. As the air absorbed the tenting reappeared, being marked at seven days, and at the end of ten days the tenting was apparently no different than previous to the air introduction.

This, of course, does not prove that the tenting seen on the screen and plate of this and other cases was not due to adhesions, as light adhesions could easily separate without pain or without increasing the irregularity. But it is improbable that they would separate after so small a quantity of air and before other non-adherent air

containing parts of the lung had shown some sign of collapse. Furthermore, it is not likely they would reorganize so firmly after separation in a matter of a week as in the above case and produce a tenting of identically the same character and extent as the original one. Moreover an irregularity due to pseudo-adhesions would be expected to reappear at the same site after restoration of the previous intrathoracic negative pressure. This occurred, as a matter of fact, in every case of pseudo-adhesions. It is therefore not thought that pleurodiaphragmatic adhesions were present in the above case, but that the negative intrathoracic pressure sucking the diaphragm to the irregular basal surface of the lung was responsible for the irregularity seen.

The principles concerned in the production of the lung dimple upon which diaphragm irregularities of the pseudo-adhesion type depend were tested out experimentally.

A bell jar was fitted with an inlet and an outlet at the top. In the inlet there was a rubber stopper through which passed a glass tube from which a pair of sheep's lungs was suspended by slipping the trachea over the glass tube and holding it securely in position by ligation. There was a stop-cock in the outlet through which the air in the bell jar could be exhausted by suction.

The base of the jar had two diaphragms, one of thin rubber three inches from the base held in position by means of a wooden hoop and sealed air-tight. The second diaphragm, also air-tight, was at the base and was made of heavy rubber, in the center of which was a lug by means of which traction could be made downward, thus pulling the upper diaphragm down by suction.

For the purpose of rendering a bronchus rigid a fine steel wire with a barb at the end was passed through the inlet down through the trachea into one of the finer bronchi and fixed by imbedding the barb in the wall of the bronchus.

As the air in the bell jar was exhausted, air from the outside passed through the inlet into the lungs, which expanded regularly; the barbed wire, not being fixed above permitted the bronchus to stretch as usual, the wire being pulled downward as the lung expanded. After the lungs partially expanded the barbed wire was

fixed at the inlet and the lungs finally expanded by further suction. Added expansion took place everywhere except over the site of the bronchus fixed by means of the barbed wire. This dimpling was increased and diminished by manipulation of the diaphragm stimulating inspiration and expiration.

It was proved that the size of the dimple depended upon whether the barb was imbedded remotely from the finest ramifications, thus fixing a large area of lung surface, served by that portion of the bronchus which came off distal to the barb and producing a large dimple, or if the barb was imbedded in a terminal bronchus the dimple was correspondingly smaller.

It is appreciated that these experiments do not reproduce conditions thought to be responsible for dimpling of the lung. Nevertheless, they furnish material support for the theory advanced.

It was hoped by means of the upper thin diaphragm to reproduce the tenting of the diaghragm. However, the distribution of the bronchi and the character of the basal surface of the sheep's lungs did not permit producing a dimple at a point where it was possible for the rubber to fit itself into the dimple.

Further experiments are in progress and will be reported later.

Conclusions.

1. The assumption that tenting of the diaphragm always diagnosticates pleurodiaphragmatic adhesions is unjustifiable.

2. Tentings of the diaphragm, frequently seen on plate and screen at the termination of the hilidiaphragm superfices with characteristics, which are associated with pseudo-adhesions, are purely phenomena brought about by two factors. (1). During inspiration the diminished elasticity or rigidity of the lower bronchial branches prevents descent of that area of the lung base in immediate relationship to the bronchus involved; thus a rimple effect is produced on the surface of the base of the lung. (2). The diaphragm being molded to the base of the lung by a negative intrathoracic pressure is held by suction in the dimple above referred to, producing a tented appearance (pseudo-adhesion).

3. Diaphragm irregularities of the pseudo-adhesion type are commonly found during the course of acute and chronic respiratory infections. In acute respiratory infections with peribronchial infiltration involving the lower bronchial branches the irregularity disappears with *restitutio ad intergrum.* In chronic, proliferative changes in the lower bronchila ramifications, the irregularity appears to be permanent in nature.

4. It is reasonable to suppose that should infiltrative or proliferative changes incident to infection not halt at a point in the bronchial tree proximal to its termination, but instead proceed to the pleura, then the diaphragm would become adherent and the pseudo-adhesion would be converted into a true adhesion.

It is hoped that this contribution will excite interest and further study of the subject.

DISCUSSION.

Dr. Hawes: I think Dr. Mattson's paper would have been strengthened if he had mentioned the effect on the diaphragm of the bronchial infiltrations. I take it the method of producing that must be the same. When the boys were coming home in considerable numbers, I was directed to examine each case in the several base hospitals, and we found repeatedly just such cases as he speaks of.

Dr. Wood: To give you an artificial pneumothorax, I had an x-ray taken and found quite marked tenting in one case, and expected to find adhesions. After the first treatment the tenting was entirely gone.

Dr. Forster: I think Dr. Mattson's very excellent paper ought to invite a great deal of attention. Of course we always think about the negative pressure in the pleural cavity, and we often wonder just what it does when the lung begins to contract. In young subjects we see so frequently not only the changes of position of the diaphragm, but the sinking in of the chest, and it is naturally one of nature's wise provosions that adhesions do occur, because they help to fix the lung and keep it properly, but not only in regard to the service of the diaphragm, not only that aspect of the lung, but this dimpling must also occur over other portions of the cavity. It cannot have any effect like it does with the dimpling of the diaphragm, but it nevertheless must occur.

Dr. Mattson: I have only one further comment to make. We had observed what Dr. Hawes speaks of. That attracted our attention, but we had no other opportunity to study the cases. The only case I had the opportunity to observe there in these particular cases, was the one I showed.

INTESTINAL TUBERCULOSIS

A Few Observations, and a Report of Cases.

By D. A. STEWART, M.D.

NINETTE, MANITOBA.

Our medical generation still regards tuberculosis as chiefly and usually a disease of the lungs, with manifestations in the digestive system, in the genito-urinary tract, in serous membranes, in bones and joints, classed as complications of that lung disease. We seldom describe tuberculous disease as general or disseminated. Our eyes are on the chest in diagnosis and in treatment. We class our cases still according to fractions of lungs involved, considering all other area diseased as added unto them.

Perhaps we should raise the complications from subordinate places to an equal or almost equal partnership in crime. Why must it always be pulmonary tuberculosis complicated by intestinal tuberculosis? Is it not sometimes rather intestinal tuberculosis complicated by disease in the lungs? Is not the combination sometimes really "Intestinal and Company" rather than "Pulmonary and Company"?

Perhaps we have underestimated the share of intestinal disease in the partnership because we have not realized how common or how early was its association with the firm.

Looking backward over what experience we may have had in the treatment of tuberculous patients, considering the frequency, the early appearance, and the persistency of intestinal symptoms; considering the old dicta, that the road to recovery lies through the stomach; that the best friend of the consumptive is a good digestion; the poor eaters do badly; that digestion is the keystone of the prognostic arch; that a flux of the bowels tends to a fatal outcome, should we not presume a much earlier beginning in the alimentary tract? Have we not abundant clinical evidence for so doing?

Post mortem findings and the observations of surgeons at operation confirm the early appearance and wide spread of the disease

in the bowel, often without marked symptoms. Heller and Wagner found twenty-eight foci evidently primary, and healed, in six hundred autopsies. Such foci are said to be not rare in the bodies of children. Opie states that in 25% of all autopsies on British soldiers nodes showing healed ulcers of the bowel were found.

Such disease must be primary. Is the intestine possibly the chief point of entrance of tuberculous disease?

There are many observations to prove definite ulceration in from 75 to 90 per cent of the bodies of those who have died of pulmonary tuberculosis.

We have, then, clinical and pathological evidence of a frequent, early and close association of intestinal with pulmonary disease, even a suggestion that the intestinal may be the earlier of the two, the senior partner. We know it is often in late stages the dominant partner.

If tuberculous ulceration of the bowel has recognizable early stages; if, by clinical observation, clinical study and clinical instinct; by opaque meals and enemata, and perhaps by other methods yet to be devised we can diagnose it at any early stage, and institute treatment at an early stage, should we not expect to better very greatly the prognosis not only of this one phase of the disease, but of the whole general disease tuberculosis as well? Early treatment will anticipate symptoms and aim not only to relieve and palliate, but to cure.

At the present time diagnosis is of greater moment even than treatment. A diagnosis well made is treatment well begun. An ounce of early diagnosis is worth many pounds of attempted cure. The gross signs we have associated with intestinal tuberculosis, pain, anorexia, nausea, diarrhoea, wasting, correspond to moist cavernous rales, night sweats and hectic flush in pulmonary disease; they are the late and prognostic signs, not the early and diagnostic signs.

Perhaps the most useful early symptom will be found to be just impaired appetite. Even abnormal appetite may be suspected.

Whatever may be said of patients in general, when the tuberculous are under consideration, a close checking up of signs and symptoms and a barium meal should be a matter of routine in the presence of any digestive upset whatever in patients of any stage,

and a routine for all the more advanced cases with or without special complaint or suspicion. If we could only read it as well, a barium meal or enema should be perhaps as common a diagnostic measure as a pair of chest plates.

Treatment has many elements. The general good conditions, rest, regulation of energy expenditure, fresh air and various other phases of the routine of a tuberculous patient, become doubly insistent, when the least or earliest sign or even suspicion of intestinal disease arises. Dietitic considerations are rather negative than positive, the providing of food not irritating or gas producing. Drugs of almost every kind have been used, antiseptics, sedatives, astringents, opiates, and even lubricants, but for the relief of symptoms rather than cure of disease.

Surgical treatment of some forms of intestinal tuberculosis is not new. Hypertrophic conditions, stricture of the small bowel due to ulceration, tuberculous disease in the appendix have long been submitted to operation, but reaction or exclusion of an ulcer-studded segment of a tuberculous bowel in order, by putting it at rest, to promote healing, is a comparatively new application of surgery.

The choice of operation is made less perhaps by the state of the bowel and more by the condition of the patient. The more complete operations seem preferable, the choice being, apparently, first excision, then exclusion, bilateral or unilateral. When these are not possible, opening the large bowel and forming an artificial anus, or even the removal of a troublesome appendix are palliative and sometimes of more permanent benefit.

The oldest of therapeutic agents has become the most modern also, something not new under the sun, light. Artificial sunlight, is applied easier, with less risk, more regularly and to patients more ill than the sun's rays. The Alpine Lamp seems to give results in cases of intestinal tuberculosis. There is no doubt as to the enthusiastic belief of the patients in its efficacy—and even that is of value.

Choice of treatment should be eclectic. All that may be of use in routine, diet or drugs, for cure or relief, should be applied, and light therapy used and persevered in in every case not definitely hopeless. Though still, perhaps, somewhat empirical, apparent results are such that patients with definite or even suspected intest-

inal tuberculosis should not be denied light therapy. So far the choice is comparatively easy. The big question is as to the usefulness of surgery; the great decision, to operate or not to operate.

The surgeon specifies that the disease should be early and localized. Especially must the lung foci be limited in extent and activity, and the small bowel not too widely involved. His ideal case has lungs slightly affected or slightly active, general condition good, and early intestinal lesion localized in right colon.

But we cannot give the surgeon all he demands. We are privileged to see the earlier stages of disease in a very few of our patients. Intestinal disease when we see it first, is, in most cases, a part of a distressful far-advanced, long-neglected condition for which not we but ignorance in and out of the profession are responsible. Surgery has little to offer if it can help only an ideal early case; if it can pluck no other brands from the burning.

Widespread active or progressive lung disease, widespread disease of the small bowel, which, however, cannot be determined before the abdomen is opened, and a poor general condition, are all against operation.

Operation should perhaps be considered when the intestinal disease is limited, especially if right-sided. In acutely progressive intestinal disease, even if widespread, the chance of operation should be taken if the lung condition is not too active. No other treatment will overtake the disease. It is surgery or nothing. In a chronic type of case also with slowly progressive intestinal disease surgery should be considered if the general condition is still good and the lung disease not active.

Over against the shock of operation, there are the compensating advantages of eliminating an active focus of disease; of clearing up the diarrhoea and of the relief of pain. Operation may be indicated even for the relief of symptoms.

Our investigation of intestinal tuberculosis at the Manitoba Sanatorium has grown in interest as it has improved in method. In the modern period of that investigation, covering a little over two years, the sanatorium has admitted about eight hundred and fifty patients and has had a daily average population of about two hundred and thirty. Of the patients under treatment at any one

time one-third only are of the ambulant type, and two-thirds infirmary cases, some of good prognosis, some hopelessly ill on admission. This was our hunting ground for intestinal tuberculosis. I have no conclusions to offer as to either diagnosis or treatment, and indeed no carefully worked over material. But perhaps a partial analysis may give an idea at least of the occurrence of symptoms and signs which raise the question of intestinal tuberculosis.

On at least a suggestion of intestinal tuberculosis one hundred and ninety-seven cases were investigated along clinical lines and by barium meals and enemata during the two years. These were classed as—

Negative	73
Doubtful—Questionable	21
Probable	18
Positive	85

Of the doubtful classes some had slight symptoms of longer or shorter duration; some more marked symptoms not quite typical. In some, plates only were suspicious; in others, both symptoms and plates. The lines were hard to draw. I am inclined to think that a fair proportion would, if the diagnostic line could be exactly drawn, fall among the positive cases. Most were in any case treated as though positive. All had pulmonary disease, mostly advanced.

Of the thirty-nine classed as questionable or probable, seventeen have made very fair improvement in intestinal symptoms, all but four of these on lamp treatment. Eight have made a little improvement, six of these on the lamp. Fourteen have done badly, mostly without lamp and mostly on account of disease other than intestinal.

Of the eighty-five positive cases, thirty-nine were on admission practically hopeless, the intestinal condition being a part of advanced general tuberculosis. Few of these were submitted to definite treatment except for the relief of symptoms.

The remaining forty-six, classed as having definite intestinal tuberculosis, had most of them also advanced pulmonary disease. Seventeen have done well and are doing well, on the lamp. Fourteen, all on the lamp, made some improvement. Nine, most of them on the lamp, did not improve. Six became worse.

Of the forty-six, four were submitted to operation. One who

had unilateral exclusion died on the tenth day after operation, with vomiting as a marked symptom. Not unlikely the death was due to operation. Two in whom prognosis was bad have done well following unilateral exclusion. One sent for operation showed so extensive disease above and below the valve that only a troublesome looking appendix was removed. He is improving steadily and is the picture of health.

Of the whole series forty-two have been on lamp treatment long enough to justify some statements at least as to general condition and symptoms.

General condition was improved in sixty per cent; not improved in twenty-eight per cent and became worse in twelve per cent.

Impaired appetite was improved in thirty-one out of thirty-nine. Pain and discomfort became less in twenty-three out of thirty-two, bowel movements, which had been abnormal in twenty-nine, were improved in twenty-one, and nausea and vomiting, complained of by eight, were relieved in four.

All these patients of course were on the general routine appropriate to their condition and had, besides light therapy or operation, whatever other treatment seemed indicated.

THE LYMPHOCYTIC INDEX IN TUBERCULOSIS.

By GERALD B. WEBB, M.D.

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The fact that lymphocytosis accompanies human tuberculosis is by no means a new observation. Neither is it a recent discovery that the higher the lymphocytosis the better the prognosis. But since these facts have not received the general recognition their clinical importance justifies we wish to further emphasize them at this time.

In 1905 Ullom and Craig[1] stated it as their impression that the actual increase in lymphocytes correspond to increased resistance of the patient to tuberculosis. In 1909[2] one of us reported a series of 500 differential blood counts on about 200 tuberculous patients of various types which definitely confirmed this impression. The average percentage of lymphocytes (lymphocytic index) was given as follows:

I.	Normal Adults	43.6
II.	Arrested or cured cases of T.B. .	41.7
III.	Improving cases	38.5
IV.	Stationary cases	34.4
V.	Cases progressing unfavorably .	27.5

It should be noted that these percentages include all the mononuclear basophilic cells, and that these observations were made at an altitude of 6,000 feet. The mononuclear percentage at sea level in normal adults is about 34. The fact that there is an absolute increase in the lymphocytes in the blood as an effect of altitude was first established by the work of Webb and Williams in 1909[3] and later confirmed by Staübli[4], Baer and Engelsman[5], Gilbert[6] and Staines and James[7].

In 1911 numerous authors including Solis-Cohen and Strickler[8], Watkins[9], Wright and King[10] and Miller and Reed[11] concluded that the percentage of lymphocytes corresponds closely to the condition

of the patient and the progress or activity of the disease and does not depend on the extent of the disease.

Murphy[12] and Taylor by x-raying mice to reduce their lymphocytes have rendered them more susceptible to tuberculosis. Morton has confirmed this work using guinea pigs and human strains of tubercle bacilli. When Murphy increased the lymphocytes in mice by immunizing against and then inoculating them with cancer, and by means of dry heat, he found their resistance to tuberculosis was greatly enhanced.

Experiments performed by Scholz[13] on the effect of recent tuberculous infection on the leucocytes in cattle, rabbits and guinea pigs showed a decrease in polymorphonuclears and an increase in lymphocytes and eosinophiles. In our work on human tuberculosis we have failed to find any definite change in the number of eosinophiles. Calmette[14] speaks of three distinct leucocytic formulae in the various types or stages of tuberculosis.

1. The "formula of resistance" found in benign fibroid cases with slow evolution and consisting of a moderate leucocytosis of not over 10,000 per cm. and a pronounced lymphocytosis with a slight eosinophilia.

2. The "formula of defense" found in cases of massive infection or special virulence and consisting of a hyperleucocytosis and abundant polynucleosis, with a constant dimunition of eosinophiles and lymphocytes.

3. The "formula of disaster" found in open cases progressing to serious caseation and breaking down with formation of cavities and consisting of a marked hyper-leucocytosis varying from 16,000 to 20,000 with polymorphonuclears around 90% and with lymphocytes and mononuclears diminished in numbers and sometimes degenerated while the eosinophiles are almost absent or broken up and fragmented.

Lymphocytosis has been reported in other conditions, such as thyroid disease and whooping cough. Daland[15] has called attention to the fact that a small cell lymphocytosis especially when accompanied by a leucopenia may be a diagnostic sign of chronic periapical dental infection in adults. We have noticed this in several cases where the teeth were in such condition as to warrant their complete

removal. Kaufman[16] states that a lymphocytosis is found with remarkable frequency in chronic disorders of the digestive tract but we have seen no sign of this. Sprunt and Evans[17] have noticed in certain acute infections a slight increase of large mononuclear and transitional cells with many pathologic lymphoid forms which is later followed by a leucocytosis largely due to increase in lymphoid cells.

Bergel[18] in a recent monograph on lymphocytosis, after noting the favorable significance of a high mononuclear count in the blood in tuberculosis and its occurrence as an early sign of improvement observes that the same thing holds good in the local lesions of the disease those having an acute course and unfavorable prognosis like the tubercle of general milary tuberculosis showing few lymphocytes while the milder and more chronic lesions are rich in them. He attributes a diagnostic value to high blood lymphocytosis as well, in that it usually indicates a pure tuberculosis, free from mixed infection. He considers the lymphocytic increase a sign of natural defensive reaction against the tubercle bacillus and believes that its value lies in the power of the mononuclear to destroy the tubercle bacillus by means of lipolytic ferments.

For the past ten years we have made routine differential blood counts on practically all of our tuberculous patients. We have become more and more impressed with the value of this lymphocytic percentage or index as a simple and accurate measure of the condition and prognosis of the patient. While such other laboratory tests as the complement fixation for tuberculosis and the Arneth index have gradually passed into the discard this simple determination has become of greater clinical importance. In order to crystallize our results we have undertaken to analyze over 200 successive counts on some four hundred tuberculous patients in all stages of the disease. The results may be very briefly summarized as follows:

Average lymphocytic index in

I.	100 Adult normals	43.
II.	130 Arrested or Apparently cured cases	45.
III.	120 Cases improving	37.
IV.	100 Cases failing	24.
V.	40 Cases progressing to death . . .	16.

The average number of mononuclears per cm. in class I was 3,354, in class II was 3,420 and in class V was 2,440. Whenever this lymphocytic index remains below 20 the outlook is practically hopeless. Of the patients whose index persisted below 20, 58% are dead, 29% are confined to bed, 10% are able to be up and about while only 3% are able to work part time. A steady or gradual fall in the index is of grave significance. A gradual rise, on the other hand, is a most encouraging sign. Of the patients whose index persisted above 40, only 2% have died, 7% are confined to bed, 20% are up and about, while 71% are leading a normal life. Those with the index over 30 have the balance of power on their side while those with an index below 30 have a doubtful outlook and prognosis.

In children the lymphocytes are much more numerous than adults. Normally the lymphocytic index is highest during the first two years of life, then falls slowly until about the age of eleven or twelve when it tends to approach the adult. At this altitude the normal lymphocytic index runs about 80 at one year of age, 70 at three years, 60 at six years, 55 at eight years and 45 at twelve years.

In analyzing our counts on children one fact was strikingly evident. Children who had recovered from tuberculosis in any form had a remarkably high lymphocytic index averaging about 56 even when the age was from 12 to 16.

These high indices did not fall as long as the children remained in good health. Children who had a mild relatively inactive glandular or pulmonary tuberculosis from which they were recovering also have a high index.

Our observations convince us that children whose index is below normal for their age require further investigation, including x-ray, as to the possibility of active or latent tuberculosis. They should be treated with rest, fresh air and good food as other tuberculous suspects, at least until the index reaches normal.

Bergel classes all the mononuclears of the blood under the name of lymphocytes on the ground that, although they are derived from a number of sources they are closely allied functionally: he even asserts that the difference in appearance between the large mononuclear phagocytes and lymphocytes is merely a matter of functional state and that one can change into the other. In this he is in agree-

ment with some European and American investigators, as Calmette and Hussey[19]. On the other hand the work of Mallory[20], Permar[21], Foot[22], and McJunkin seems to have established rather securely that the large mononuclear has a special origin from the vascular endothelium and a very special functional capacity for ingesting solid foreign particles, which, according to this school, is not shared by those cells which are certainly derived from lymphoid tissue. This large endothelial leucocyte which includes the so-called "Transitional" cell is also the cell chiefly concerned in the local lesions of tuberculosis, syphilis and typhoid and may reach 15% in the peripheral blood in malaria. McJunkin[23] has devised a method of identification for these endothelial leucocytes by the use of a benzidin-polychrome stain. He reports that these calls are increased in advanced tuberculosis in guinea pigs up to 10%, whereas there are only 1.5% in normal pigs. When we went over our counts with this in mind we found in the far advanced active cases an average of 6% endothelial leucocytes or 840 per cmm., with 16% total mononuclear elements or 2,440 per cmm. It should be especially noted that the average total number of leucocytes was 14,000.

In the arrested or cured cases we find an average of 8% endothelial leucocytes or 608 per cmm. with 45% total mononuclear elements or 3,420 per cmm. Total leucocytes averaged 7,600.

In the normal cases we find 8% endothelial leucocytes or 624 per cmm. with 43% total mononuclear or 3,354 per cmm. Total leucocytes in this series averaged 7,900.

Although far advanced cases show a decreased percentage of endothelial leucocytes there is an absolute increase because of the leucocytosis present. On the other hand these far advanced cases have a very marked decrease in the absolute and relative numbers of lymphocytes present.

These findings, with others already cited, would certainly seem to indicate that the lymphocytes, and not the endothelial leucocytes, are the important factors in measuring resistance to tuberculosis in human beings.

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INFLUENCE OF VARIOUS SYMPTOMS IN THE PROGNOSIS OF PULMONARY TUBERCULOSIS.

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In two previous communications I have attempted to analyze the relative resultant effect of the presence, absence and location of rales, together with the importance during treatment of their increase and decrease, in the prognosis of pulmonary tuberculosis.

The purpose of this paper is to determine the comparative value of certain symptoms in aiding us to predict for our patients something of their ultimate chances of recovery. This analysis, as in the two previous communications above mentioned, is based on a study of one thousand consecutive admissions to the Trudeau Sanatorium during the years of 1907 to 1913. Twenty of these cases for one reason or another have not been included in these statistics, so the tabulations are based on 980 cases only. Due to our follow-up system employed at the sanatorium, we are able to tell the present condition, or to be more exact the condition in 1918 of all but less than 2% of these 980 cases.

The terms "well," "living," "dead" and "unknown" have been used to describe the state of health of these 980 patients in 1918, or from five to eleven years after discharge; "well" referring to those patients who have been apparently well and working for a period of at least two years, "living" including either the relapsed cases or those of whom we can get no further information except the fact that they are still alive.

Mode of Onset.

The first point to be considered is the mode of onset of the disease in these 980 cases, and the significance of its various types as regards prognosis. In considering the five most common types of onset, namely, the catarrhal, the insidious, the pleuritic, the haemoptoic and the febrile, we note from the following table that

the way the disease began played little or no part in determining its future course. In the five types of onset above mentioned the "wells" only vary from 58% to 68%, while the deaths vary from 20% to 27%.

	Onset Type		Condition in 1918							
			Well		Living		Dead		Unknown	
	No.	%	No.	%	No.	%	No.	%	No.	%
Catarrhal	473	48.2	289	61	70	15	110	23	4	1
Insidious Gradual	350	35.71	237	68	38	11	70	20	5	1
Pleuritic	72	7.34	42	58	14	19	15	21	1	2
Haemoptoic	50	5.1	33	66	5	10	12	24	—	—
Febrile	15	1.5	9	60	2	13	4	27	—	—
Gastric, Dispeptic, Indigestion	8	.8	7	88	—	—	—	—	1	12
Glandular	5	.5	3	60	1	20	1	20	—	—
Family History plus T'b'r'n	3	.3	1	33	1	33	1	33	—	—
Fistula in Ano	2	.2	2	100	—	—	—	—	—	—
Pneumonia	2	.2	1	50	—	—	1	50	—	—
Total	980	100	624		131		214		11	

Sputum.

Among our 980 cases 591 or 60% showed tubercle bacilli in their sputum, 464 of these while in the sanatorium and 127 before coming to us. The grave importance of these findings as regards prognosis is clearly shown when we realize that only 53% of these cases where the sputum was positive are well and working, as contrasted with 79% among the negative cases, while the mortality among the positive cases, from five to eleven years after discharge, is 32% as compared to only 6% in the negative cases.

Sputum.			Condition in 1918							
			Well		Living		Dead		Unknown	
Type	No.	%	No.	%	No.	%	No.	%	No.	%
Negative	389	39.7	307	79	51	13	24	6	7	2
Positive	591	60.3	318	53	79	14	190	32	4	1
Total	980	100	625		130		214		11	

Haemoptysis.

Under the heading of haemoptysis we have arbitrarily set the amount of one teaspoonful or more of blood as constituting a definite haemorrhage. Doubtless most of us are apt to consider this symptom as being of rather grave prognostic value in comparison to

the absence of haemoptysis. The figures in the following table are rather surprising, for 61% of the haemoptoic cases are well and working as compared to 65% of our non-bleeding patients, while the death rate is practically the same for the two types of cases; namely, 23% and 22%.

Haemorrhage.			Condition in 1918							
			Well		Living		Dead		Unknown	
Type	No.	%	No.	%	No.	%	No.	%	No.	%
Negative	636	64.9	415	65	77	12	136	22	8	1
Positive	344	35.1	210	61	53	15	78	23	3	1
Total	980	100	625		130		214		11	

Weight.

The following table will illustrate the beneficial import of a gain in weight among our tuberculosis patients. Over 67% of our series showed a gain in weight averaging 8½ pounds per capita, as contrasted to only 28% who lost weight, and approximately 5% whose weight remained stationary. Among those who gained, the figures for the "wells" and "deads" are 67% and 20% respectively, contrasted with 57% and 27% of those who lost weight. The forty-seven cases whose weight remained stationary did practically no better than those cases who showed a loss of weight.

Weight.			Condition in 1918							
			Well		Living		Dead		Unknown	
Type	No.	%	No.	%	No.	%	No.	%	No.	%
Gain	657	67.04	441	67	81	12	127	20	8	1
Loss	276	28.16	157	57	42	15	75	27	2	1
Stationary	47	4.79	27	57	7	15	12	26	1	2
Total	980	100%	625		130		214		11	

Pulse.

The symptomatic importance in prognosis of the pulse is emphasized by the following table. Cases are rated as "pulse positive" in this series having a pulse rate of 90 or over for five consecutive days where diagnosis fails to reveal any other cause than tuberculosis. 69% of the negative pulse cases are well and working as compared to only 49% of those having tachycardia, while only 16% of the former class are dead as compared to 38% of the latter. The

grave prognostic significance of rapid pulse is undoubtedly largely due to the fact that it is so often accompanied by fever, which, as we shall see in the next table, is the gravest of all symptoms which we have studied.

Pulse.			Condition in 1918							
			Well		Living		Dead		Unknown	
Type	No.	%	No.	%	No.	%	No.	%	No.	%
Negative .	717	73.1	496	69	97	14	114	16	10	1
Positive . .	263	26.9	129	49	33	13	100	38	1	—
Total .	980	100%	625		130		214		11	

Fever.

Cases are here classified as "fever positive" where the patient ran a temperature of 99.5% or over for five consecutive days, which could not be explained by any other cause than tuberculosis. By reference to the following figures it will be noted that the percentage of "deads" among our febrile patients is nearly as great as the percentage of "wells"; namely, 43% and 45% respectively, while of our febrile patients 68% were well and working five to eleven years after discharge, and only 17% had died.

Fever.			Condition in 1918							
			Well		Living		Dead		Unknown	
Type	No.	%	No.	%	No.	%	No.	%	No.	%
Negative .	809	82.6	548	68	110	14	141	17	10	1
Positive . .	171	17.4	77	45	20	12	73	43	1	—
Total .	980	100%	625		130		214		11	

Although not strictly classified as symptoms according to the title of this paper, it might be of interest to consider from a prognostic standpoint the influence of sex, family history and the stage of the disease on admittance to the institution.

Sex.

The following table discloses that the proportion of males to females entering the institution is very nearly equal, as is the percentage of "wells" among the two sexes, namely, 63% and 65% respectively; the males, however, have slightly the higher death rate in our series, with 25% as compared to 18% of the females.

Sex.			Condition in 1918							
			Well		Living		Dead		Unknown	
Type	No.	%	No.	%	No.	%	No.	%	No.	%
Female . .	458	46.73	297	65	75	16	80	18	6	1
Male . .	522	53.26	329	63	55	11	133	25	5	1
Total .	980	100%	626		130		213		11	

Family History.

In considering family history in this paper we have reference only to the immediate family; namely, father, mother, brothers or sisters. It is of interest to note that 30% of our cases have a positive family history, yet this fact seems not to have any prognostic value, as a study of the table reveals only a small variation in outcome as between our positive and negative family history cases.

Family History.			Condition in 1918							
			Well		Living		Dead		Unknown	
Type	No.	%	No.	%	No.	%	No.	%	No.	%
Negative .	690	70.4	441	64	90	13	153	22	6	1
Positive . .	290	29.6	184	63	40	14	61	21	5	2
Total .	980	100%	625		130		214		11	

Stage of Disease on Admittance.

The following table is indeed encouraging, for over 78% of our incipient cases have resumed normal life, while less than 10% have died. Even among the moderately advanced cases over 70% are alive, although only 57% have been restored to good enough health to allow them to work. It seems indeed gratifying that there are 25% of "wells" among our far advanced cases.

Stage of Disease on Admittance.			Condition in 1918							
			Well		Living		Dead		Unknown	
Type	No.	%	No.	%	No.	%	No.	%	No.	%
Incipient .	352	35.91	275	78	43	12	28	8	6	2
Moderately Advanced .	612	62.44	350	57	84	14	173	28	5	1
Far Advanced	16	1.62	4	25	3	19	9	56	—	—
Total .	980	100%	629		130		210		11	

In closing, it might be interesting to note the present condition of these 980 cases, regardless of any symptoms, except the fact that they were patients in the sanatorium. 214 of these are dead and eleven unknown. Of the deaths, 180, or 84%, were caused by tuberculosis and 34 from other causes. So that of 980 cases whose condition has warranted their admittance to the sanatorium 75%

are well or living from five to eleven years after discharge, while practically two out of every three of them have been restored to earning capacity.

TOTAL RESULT.

	No.	%
Well	625	64
Living	130	13
Dead	214	22
Unknown	11	1
	980	100

DISCUSSION.

DR. DARLINGTON: I would like to say that I think this is one of the most important papers before this Society. If we could have more of this class it would be a wonderful thing.

DR. FORSTER: I always enjoy a paper of this sort, which calls attention to the wonderful record that Dr. Trudeau keeps of his discharged cases. I note that Dr. Trudeau has chosen a temperature above 99.5 and a pulse above 90—I mean by that tuberculosis poison manifests itself in the pulse and temperature at a point below that. I understand Dr. Trudeau's reason for using those two two points, but I think it will be well for him to emphasize the fact that they are arbitrarily chosen and do not represent the point at which the tuberculosis poison manifests itself either in pulse or temperature.

DR. HAWES: I am glad to see that Dr. Trudeau called attention to a rapid pulse. In Massachusetts, in private practice, patients are coming in more and more with a good record of pulse taken. Sometimes, however, I send many of my patients to the hospital to have pulse and temperature taken for a week or ten days. They come back with a record nicely put down, but rarely of pulse.

I am very glad that Dr. Trudeau called attention and gave some figures in regard to the pulse.

A MEMBER: In addition to the clinic that Dr. Trudeau brought out, there is a group of social psychology. Some years ago I tried to work out a formula that might apply on the education, adaptability and co-operation on the part of the patient. I think those factors play a very important part in the prognosis of tuberculosis, because it is difficult to estimate it or put it down quantitatively, but I think Dr. Miner, in his many years' experience on psychology of tuberculosis, might offer some very valuable suggestions.

DR. PROBST: What is the natural pulse? I remember very well a family in which there was a mother and three daughters, one of the daughters having tuberculosis. Her pulse was above 90 oftentimes, and I commented on it, and the mother said, "It always has been that way; mine is, and so is the other sister." I tried them, and they were running above 90.

What is the natural pulse?

DR. MINER: The interesting question of pulse. We all recognize, with

Dr. Probst, that there are people who have faster pulse than others. My pulse, before I was a sick man, ran faster than the average man's.

I think Dr. Trudeau has chosen his point very wisely. However, Dr. Trudeau could not take into consideration every possible factor. The effect of the psychology of the patient upon the prognosis is enormous. None of us doubt that. The means of recording that and observing it are very difficult. The things Dr. Trudeau took up are things he could get a definite record of. It would be very interesting to study the psychology of various patients. You know that some patients come to you with not very much trouble, and you know they have not enough stuff in them to get well. You see others with very bad trouble, and you study their psychology, and you know they will fight it through in spite of the devil. But to put that down in statistics is an extremely difficult thing, therefore, while I agree with Dr. Probst that it is a valuable factor, I doubt if Dr. Trudeau would have brought it in his summary.

We always feel envious of Saranac men of keeping such statistics. Our own private patients cannot do it. It is a great regret to me that I cannot keep records like Dr. Trudeau, and I think the country is indebted to the Trudeau Sanatorium for the careful way they have followed their cases. They have followed them carefully, and in the long run these analysis they give from the results they are getting to be valuable to us.

Dr. Wood: The fact that haemoptosis causes so much fear and upset, it is comforting to the physician to find that it is not as dangerous a thing as high pulse, as there is nothing that patients are so much afraid of as any such signs.

Dr. Brown: Dr. Trudeau is making these very interesting studies from year to year, and I think we ought to bear in mind somewhat the type of cases he is studying. The patients admitted to the Trudeau Sanatorium are carefully selected cases, early cases as a rule, and some of the data I understand he gives relates to the study of the first week of the patient's admission. That means, when he speaks of haemoptosis—I think that may mean a very different thing than if the patient has continuous or frequent haemoptosis from time to time, and the prognosis would be very different in the two types of cases. The figure of the pulse at 90 adopted for men at Trudeau—they try to decide when a case was active or inactive. We felt if a man had a pulse over 90 that it was due to some poisoning of tuberculosis bacillus, and was an active case, though we have had to bear in mind some other points.

I recall some years ago, when I was examining patients for admission to a hospital, we had some who came to the examining office and never had a pulse less than 140, and one woman in particular we thought she had tuberculosis. She said at home she had 80 to 90, and we admitted her and found that that was her true pulse, and a pulse taken in a doctor's office very often does not give an accurate index of what a patient's pulse is.

In regard to the temperature, we consider over 99 three or four times a week an indication of activity in a man, and 99.6 in a woman. All of those points have to be taken into consideration in the various factors Dr. Trudeau mentioned in his very interesting paper.

JUVENILE TUBERCULOSIS.

By JOHN B. HAWES 2ND, M.D.,

BOSTON, MASS.

The diagnosis of pulmonary tuberculosis in the adult at the present time is in a fairly satisfactory condition and likewise, in my own state at least, the provision for treatment in sanatoria and hospitals is fairly adequate. When we come to the diagnosis and treatment of tuberculosis in children, however, we find a far different situation. Standards on which the diagnosis of bronchial gland tuberculosis may be based are conspicuous by their absence while institutions where children may receive prompt, proper and efficient treatment as yet at least are excedingly rare. It is for this reason that I have chosen this subject,—the diagnosis of juvenile tuberculosis,—believing it is to be one of the very greatest importance and hoping that what I have to say will promote free discussion.

Children with tuberculosis may be divided into three groups, as follows:

1. Those who have a tuberculous infection causing no symptoms and presenting no signs.

2. Those who have actual tuberculosis of the lung itself. Such cases are comparatively rare, easy to diagnose and require in the majority of instances prolonged hospital or sanatorium treatment.

3. Children with bronchial gland tuberculosis, causing both signs and symptoms.

I shall confine myself to this last group and shall here consider only the diagnosis of bronchial gland tuberculosis. In a recent article entitled, "The Need of Sanatorium Treatment for Tuberculous Children,"* Dr. Walter Griffin in referring to the diagnosis of tuberculosis in children uses the term "definite pulmonary signs" four times in the course of what he says but he does not state what these "definite pulmonary signs' 'are nor how to find them. My experience leads me to believe that "definite pulmonary signs" in

*Boston Medical and Surgical Journal, March 9, 1922, pp. 316-318.

bronchial gland tuberculosis are rare. Some years ago Dr. Frederic C. Shattuck, one of the greatest clinicians it has been my privilege to know, was discussing hyperthyroidism at a clinic of fourth year students at the Harvard Medical School. A certain student, of a type which is always present in medical classes, leaned forward and asked Dr. Shattuck if a certain sign was not pathognomic of this condition. Dr. Shattuck looked at him for a minute and then said as follows: "Young man, 'an evil and adulterous generation seeketh after a sign and there shall no sign be given to it.'" This, in my limited experience, applies to bronchial gland tuberculosis.

There are certain definite pulmonary signs, easily recognizable by anyone, in well advanced parenchymatous tuberculosis in children as well as in adults, the chief of which, naturally enough, is the presence of tubercle bacilli in the sputum. In tuberculosis of the bronchial glands the situtation is different. I do not believe that there is any one or two or three definite pulmonary signs of this condition. I know of no diagnosis which is based more on a general sizing up of the situation from every angle and the balance of one thing against another and the careful weighing of all the evidence pro and con than this.

Dr. Lawrasan Brown has helped us all in diagnosing pulmonary tuberculosis in adults by the formulating of his five diagnostic points. With these we are all familiar. For my own benefit I had already prepared five diagnostic points which I have found useful in the diagnosis of bronchial gland tuberculosis. I have used these points not only in the diagnosis of my own patients but for teaching purposes. These are as follows:

1. A positive skin tuberculin test unless the child has recently recovered from measles or any of the other acute infections which might lead to a negative test, or unless the child has advanced tuberculosis.

I do not see how there can be any difference of opinion concerning the need of having a positive tuberculin test of some form or other with the exceptions mentioned above before one can come to a definite conclusion concerning tuberculosis of the bronchial glands. It likewise should be remembered, however, that a positive tuberculin test alone does not necessarily mean tuberculosis of the

bronchial glands and that the diagnosis of this condition must be based on much more than this.

2. A definite history of exposure from either human or bovine source of tuberculosis.

In regard to this point, as in others entering into the diagnosis of this condition, the dictum that "absence of proof is not proof of absence" holds true. Because it cannot be shown, for instance, that a given child has been exposed to tuberculosis either human or bovine is, of course, no proof that the child is not tuberculous. On the other hand, a definite history of tuberculosis exposure is strong, confirmatory evidence that other signs and symptoms which may be present may likewise be due to tuberculosis. I have, therefore, placed this point second in my list.

3. Constitutional signs and symptoms, particularly loss of weight or failure to gain weight, along with "ease of tire" or undue fatigue, fever or rapid pulse.

The grouping of "failure to gain weight" over a period of six months along with "undue fatigue' 'or "ease of tire" I believe to be important. I have seen children in whom the diagnosis of tuberculosis of the bronchial glands has been suggested who undoubtedly have "undue fatigue" but who have been well nourished and fat, indeed rather too much so. I have seen others who have failed to gain weight or who have even lost a little weight over a period of months but whose parents have not noticed "undue fatigue" or "ease of tire." Both these types of cases have generally turned out *not* to have tuberculosis. The combination of these two conditions I believe to be the most important of the constitutional signs and symptoms. Fever and rapid pulse are caused by too many other conditions to be of particular importance in this diagnosis.

4. The presence of enlarged bronchial glands as shown by x-ray or by clinical examination of the chest.

I hesitated long before I added the phrase "as shown * * * by clinical examination of the chest." For my own purposes I should have left it out altogether because I believe that bronchial glands are rarely recognizable by clinical methods alone unless they have reached such an extreme size when the diagnosis is apparent. These latter cases are rare. I have no doubt but that there are clinicians

who can satisfy themselves that they have demonstrated parasternal and interscapular dullness as indications of enlarged bronchial glands but I confess that I have rarely been able to satisfy myself on this point. I feel the same way about these two signs as I do about Kroenig's so-called "apical isthmus of dullness." Attempts to demonstrate this I believe to be largely a waste of time.

I might well have added to my statement in regard to the x-ray the phrase, "interpreted by an expert." I recently saw a child in whose chest an enthusiastic clinician had percussed out parasternal dullness so that even I, a doubting Thomas in regard to this, was satisfied that such dullness existed. He said that it was due to enlarged bronchial glands and he triumphantly demonstrated a shadow on the x-ray plate. The shadow did not look like bronchial glands to me and I was relieved when later on the x-ray man who took the picture whispered to me that the shadow was due to an abnormal position of the great vessels and not to the enlargement of bronchial glands. Such cases, however, are rare.

5. The absence of other evident sources of infection or toxemia such as, (a) infected tonsils or adenoids, (b) carious teeth, (c) intestinal disturbances and particularly a chronic appendix, (d) other possible sources of infection such as middle-ear, glands in the neck, bronchpneumonia, measles, whooping cough, etc.

This point is an entirely negative one but none the less important. Recent experience has taught us all to how great a degree any acute infection, particularly influenza, may cause enlargement of the bronchial glands. In addition to this, an infection from the throat, notably tonsils, will undoubtedly in some way which I cannot explain skip the cervical lymphatics, lodge at the hilus of the lungs and there cause enlargement of the hilus glands. Dr. Elliott of Toronto told me of a case where the removal of a chronic appendix was at once followed by the subsidence to their normal size of the bronchial glands which prior to this had been considerably enlarged.

These, then, are the five points to which I should give most serious consideration before making a definite diagnosis of bronchial gland tuberculosis. The only one, the presence of which I should insist upon with the exceptions mentioned above, is a positive tuberculin test. In the great majority of cases likewise I believe

that there should be x-ray evidence of glandular enlargement. The other three points are important but not necessarily essential to the diagnosis.

DISCUSSION.

Dr. Lloyd: Mr. Chairman and gentlemen, I have been interested in the work that Dr. Hawes read his paper on.

In Rochester we have nearly one hundred children in our sanatarium, and we have a dispensary where we see a great many children, most of whom are contact cases, and we have been up against the problem of saying which child should go to a sanatarium and which child should stay home, and we have arrived at about the same conclusion that Dr. Hawes gave in his synopsis.

We place more importance on the contact and age of the child than any other single factor. Dr. Hawes did not give the age of his children. We feel that under five or under eight that an intimate contact almost always means infection. I say that and I know I will be jumped on, but I am talking about a child whose home conditions are bad, who are not properly clothed, and fed and who are in contact with tuberculosis, or have an infection which requires treatment. We have given these children sanatorium treatment.

Recently there has been a big tonsil drive in Rochester and there were eight thousand children operated on. Of this number, my x-ray man got a picture of two thousand before they were operated on. The conditions under which they were operated were as good as the average private operation would be, and the children were probably better examined. We are not prepared to give any report of these findings, but we have classified the children, these x-ray pictures as negative, suspicious and positive. Just what constitutes positive we do not know, but where the child is sick, clinically, whether there are any physicial signs, and whether the x-ray shows a big shadow, with shadings running toward the apex, we call that a positive case from an x-ray standpoint.

We had a very interesting autopsy a few ago days in a sixteen month old baby. This child's mother has tuberculosis, and had it ever since the birth of the child, and the child was living in the house with its mother. It was almost impossible to say what the baby was sick with, but the baby was sick. It was improperly cared for by an older sister, the feeding bad and the home conditions bad, but we took the child in and it got along bully for a while. Then we got some influenza cases, and this baby developed pneumonia and died. No physical signs of tuberculosis were there before its death. The x-ray pictures were not good. We got a post mortem picture. We found a hilus gland about as large as a pea. We cut it open and got tubercle bacilli from that gland, and as far as we could make out there is no enlargement of the hilus gland demonstrable, and we do not know where we are at.

Personally I feel that when we have these conditions fulfilled, and particularly where there is no other cause for the child's illness, we ought to call that child a tuberculous child—I do not mean that we ought to tell the parents that the child has tuberculosis but we ought to remember that a large percent of

people die with tuberculosis as they reach adult years,—there must be a time between infection, when the disease is active within the glands, and until we find some definite sign by which we can diagnose an active tuberculosis, we ought to classify them as tuberculous, and put them under the best conditions possible.

Dr. Chadwick: In regard to Dr. Hawes' paper, I will be as brief as possible and will read an extract of a paper which I read at the Sanitarium Association last spring, in regard to the classification of juvenile tuberculosis.

The diagnosis of juvenile tuberculosis is very essentially one in which the symptoms have to be considered more than physical signs. I would differ from Dr. Hawes with regard to the value of physical signs in diagnosing tuberculosis in children.

When we make a diagnosis of tuberculosis, in childhood we should then divide them into two classes, bronchial gland tuberculosis and incipient tuberculosis. The incipient tuberculosis would be a group where there were not only the glands but also a bronchial thickening with nodules along the bronchi. Then we would add to our present classification those two groups, and then we would reach the adult type, we would have the mean and the advanced. It is very important to consider symptoms, as I said before, along with the physical signs in arriving at the diagnosis, and I would like to hear some comments on this subject in regard to the classification.

The President: I will call on Dr. Otis.

Dr. Otis: I am very glad to say a few words on this subject, as there is no subject that seems to be more important than studying tuberculosis of children, and I think those who have had much to do with children would make the diagnosis very difficult for several reasons; so far as my experience goes it is very rare to find any definite physical signs in the chest of a child that would indicate active tuberculosis exists. It is hard to find a single instance in a child, if examined, in which there are any definite physical signs. The child is so susceptible that you cannot prove the enlarged glands are tubercular, judging from the other glands. In the third place nobody knows what the normal x-ray picture of a child is. What is the normal x-ray? They were unable to state what it was, even the National Tuberculosis Association, in its report. All tuberculosis is essentially a glandular disease. In a book a doctor gives over one hundred of his autopsies, and declares in every instance the infected chest and secondarily the adjacent bronchial glands were infected, and others declared they were infected first. Therefore, it is a very difficult thing to determine the absolute diagnosis of tuberculosis in children.

Then you come down to the manner of the tuberculosis test. Up to the present time we have 698 children up to the age of fourteen. We have given 250 tuberculin, and after we had obtained what we supposed was a reliable tuberculin, we began to suspect from our result that it might not be, and we wrote to the Public Health Laboratory and asked them if they had standardized tuberculin, and we are trying out half a dozen different preparations from reputable dealers to find out how much difference there is.

It seems to me that all workers with children and all children's clinics in different parts of the country should determine upon definite standards, that is, different standardized methods, and we have one and the same tuberculin, and then they could compare their results. I do not know of such combined experiments being made.

THE PRESIDENT: Dr. Elliott.

DR. ELLIOTT: I want to congratulate Dr. Hawes in his classification. His first one we must accept. We know it is a tentative test. The second one is relatively unimportant, although it is suggestive, of course. But No. 2, if one applies the tubercular test, is relatively unimportant.

If time would permit, Mr. Chairman, I would like to ask Dr. Parsons to speak of where we are following up some 700,000 children with tuberculin and other records.

I must say that again and again patients are sent to us with a suggestion of tuberculosis, because of chronic cough and physical signs in the chest.

We have seen a large number of non-tuberculous patients referred to our clinic, and there are various conditions which result; that condition we see so much of, the chronic lower lobe infection, the pneumonic process, where it is going on.

THE PRESIDENT: Dr. Parsons, will you continue the discussion?

DR. PARSONS: Mr. Chairman and gentlemen, I feel I am rather an intruder, but I will say a few words. I know Dr. Hawes' work very well.

To be brief, in our clinic, I think we have seen 5,300. We have records and have them under our observation. As far as I have gone over them, 52 per cent of contacts are tubercular. Many of these are sent to us by the schools, because they are underfed or undernourished, or they are tubercular contacts.

An interesting point is this, that has come up in connection with the indoor work, that is, the feeding of children under two years of age. In fact, the early childhood has attracted a great deal of attention all over the country. The mortality is somewhat appalling.

May I give these figures? They are not my own, but I am sure I may be permitted to state them. Of 285 children under two years of age sent in for malnutrition 85 were positively tuberculous. Under two years of age those children have been looked up lately. Of those 85, 68 were followed up, and 64 are dead, two are dying, and two are well. I think that corresponds with the experience of most of these infections under two years of age.

With regard to classification, I think it is a subject too large to take up in a few minutes. There are so many different types, first of all the children sick and children not sick. Amongst the contacts that are brought to us merely for examination, merely to find out the family's infection, we find a large number of children in perfect health and all they require is supervision, schooling, direction, and we want them with the school doctors to see that they are all right, and then comes the question of children who are sick and have symptoms of it, and we find everything to the smallest type of glandular tuberculosis. Some are pulmonary cases pure and simple, and diagnosis and x-ray count for everything.

They give you a clear picture. When you come to the pulmonary you have several important things. We have the history of infections which may or may not account for anything. We have dullness, as was suggested by one of the speakers.

The vertabrae dullness I do not feel so sure of. Possibly I do not do it properly. To confirm that you have the x-ray and the tuberculin, but do not let us forget that the complete examination of the child from top to bottom is just as important in this work; it is more important in this work to see that we are not overlooking something.

I have for a short time been looking over the surgical cases in children's hospitals, the surgical tuberculosis from the pulmonary standpoint, and today I think I can say that I have found but one child who had not a pulmonary or a bronchial evidence of the disease.

Colonel Bushnell: I am very deeply interested in this subject, and I think one of the troubles of the discussion of children's tuberculosis is that we do not make a sharp distinction between glands and inflammation. We guess at a lot of signs. We get some signs from the hilus. You have got to prove that that is not due to the inflammation that surrounds the glands. I doubt very much if you find glands large enough to find them. I am able to agree with Dr. Hawes that that distinction must be made quite carefully.

I recall a picture in a German book in which a bronchial gland was larger than the upper lobe, and it extended from the hilus—if you have a bronchial gland like that there is no question that it would give symptoms, but that is a rarety. In a majority of the cases the glands are small, and the main things are the surrounding trouble.

I want to say one word about tuberculin. I am suspecting a good deal about it, and it has been said that the tuberculin is very sensitive. There are cases in which there was a negative test and undoubted tuberculosis, and the patient was not dying, and such cases have been cited. I do not think we have reason to feel that we have exhausted our resources when we record a negative. There is a great deal to be learned in that direction.

Dr. White: I was very delighted to hear Dr. Hawes' paper. It brings up at the present time an enormous economic question in this country.

It has been pointed out that we do not get to know the normal chest, and the importance of this diagnosis must rest and have rested on the picture of the root in the x-ray production. The National Tuberculosis Association took a definite step forward last year in appropriating part of its funds to research work, in the hands of a small committee. This small committee asked three x-ray men in this country, Dr. Baetzer, Dr. Brennan and another doctor, accompanied by three clinicians, Dr. Landis, Dr. Ostrian and Dr. Blackman, as clinical examiners, to take the problem of what constituted the normal picture, of at least the x-ray picture of normal lungs, between five and ten years of age.

These men have worked with the utmost care and decision and interest, and all of them have said—their report has been read on the subject at the National Association meeting. I have no right to invade it, but they all

unanimously agreed, and they have established for the first time, I think, an x-ray picture of what constitutes a normal lung of a child between five and ten years of age. The clinical men have offered also a report of certain signs in this examination. That also I have no right to invade, but the one thing that struck me was that out of 3,000 children there was only one without clinical history of the disease of the lungs of some sort—these all leave undoubtedly traces in the lungs, a picture which must be taken into account in these reports, which are being given, and I hope that definite step forward in research work by the National Association will put us a little more firmly on our feet.

END RESULTS IN THE TREATMENT OF FAR ADVANCED PULMONARY TUBERCULOSIS BY ARTIFICIAL PNEUMOTHORAX.

By J. WOODS PRICE, M.D.,

SARANAC LAKE, N. Y.

In thinking of the treatment of far advanced pulmonary tuberculosis by means of artificial pneumothorax, we are apt to let our thoughts dwell on those comparatively few cases in which a startlingly good result is obtained within the first few months of treatment, and forget about the many pitfalls that come later—or we may even dwell upon the still fewer cases that go on to a good arrest in the satisfactory way in which they began. And yet I think to every man who uses this method of treatment to any extent, there comes a time when he asks himself if it is worth while, and whether the final state of these patients is not worse than the first.

Particularly does he feel this way when after a most encouraging few months a spontaneous pneumothorax develops, probably the most unfortunate complication that happens in the course of the treatment. I can think of no worse state in which the tuberculous patient can find himself than to have a spontaneous pneumothorax with a bronchial fistula, through which he constantly raises the overflow of pus that nearly always collects in his pleural cavity. And add to that a fistula in his side that is apt to follow some unavoidable accident in attempts to relieve him by repeated tappings of this ever thickening fluid, and you have a picture of misery indeed. It is to be hoped that, with the greater interest developing in thoracic surgery, some way will be worked out to help this type of case, many of whom have excellent resistance and go on for months in a pitiable state.

Another disappointing result is in the type of case, that in spite of your best efforts to select the proper time for refilling after the appearance of fluid with violent reaction,—I mean when the fluid comes on so rapidly and in such quantity that it becomes necessary to remove it—and when the lung re-expands under the fluid, and becomes adherent inch by inch to the diaphragm, and finally creeps up the chest wall, sticking fast as it goes and leaving you with a small pocket into which the injection of small amounts of air does

neither good nor harm, and the patient is back where he started, with constitutional symptoms and a worse cough, because of an adherent lung. These cases have already interested the surgeons and make excellent subjects for Thoraco-Plasty.

The reactivation of quiescent disease in the other lung, or the development of disease not present there before, is a complication that puts an end to the treatment, and is one for which nothing can be done other than to fall back on routine measures. I do not mean that compression should be given up altogether, but that it cannot be carried on with its former vigor.

Infection of the pleural cavity from the outside is a very infrequent result of the treatment, and is probably avoidable, and should be treated as any other empyema.

These and combinations of these are some of the end results that are very discouraging to those who make use of this treatment, and it is for fear of these results that I have only use it in those cases that seemed to me to have had no other chance for recovery.

I have spoken of these bad results, before reporting to you in detail one case in which a particularly good result was obtained, and which I am submitting as proof of what can be done. The case was selected because the patient had been in good health for ten years after the induction of artificial pneumothorax,—died of an intercurrent disease, after a short illness, which had practically no effect upon the condition of her lungs, and because we were able to obtain the specimen, which I have here to show you.

Miss X., school teacher, age 43, referred to me by Doctor E. R. Baldwin, for treatment with artificial pneumothorax, and was one of our first cases treated by this method. Beginning of infection dates back to 1901. Was one of the first patients treated at the Ray Brook Sanatorium in 1904, after which she continued teaching in the winter and spending her summer vacations in the Adirondacks until November, 1911, when she had a complete breakdown. Came to Saranac Lake at that time, and was put to bed in favorable surroundings. For four months she ran an acutely progressive course —average daily temperature 101, going at times to 103—night sweats, cough, and expectoration loaded with tubercle bacilli.

Physical examination revealed moderately advanced disease of

the right upper lobe, and far advanced disease throughout the left lung with cavity formation in the upper lobe and consolidation in the lower.

First injection March 2nd, 1912. Temperature became subnormal on day of injection, rose to normal on second day thereafter, and continued normal or 99, going only once to 100 for three months lacking a few days. During this time refills were made at regular intervals, and the pleura being free of adhesions, a practically complete collapse was obtained. At this time pleurisy with effusion with violent reaction developed. The temperature became normal again in three weeks with no other treatment than watchful waiting. Six weeks after the beginning of the pleurisy, the fluid was removed and replaced with gas. This treatment, aspirating and refilling, with the addition of an occasional injection of iodoform solution into the pleural cavity, was kept up for one year more, when after seventeen months of treatment altogether, it was discontinued because of the small pocket of gas present due to re-expansion of the lung and contraction of the pleural cavity.

By this time the patient was in excellent health, and had a negative sputum and was symptom free with the exception of a certain amount of dyspnoea and symptoms of a catarrhal bronchitis. She resumed her teaching within another year, gradually increased her activities, tutoring outside of school hours, teaching Sunday school, etc., and in summer doing practical nursing,—in short, living a normal life, or rather an abnormally active and useful one, until in February of this year it was discovered that she had double ovarian tumor. A good arrest of what seemed hopeless, far advanced pulmonary tuberculosis ten years previously, and as far as her lung condition was concerned, there seemed no reason why she might not have carried on for many years to come. Repeated examinations of the sputum in the past eight years have failed to reveal the presenc of tubercle bacilli.

On February 9, 1922, she was operated upon and two large adeno-cystomata of the ovaries were removed. Examination of these tumors showed small masses of carcinoma at the base of each tumor mass. Metasteses immediately developed, confined chiefly to the abdominal cavity, but also extending to the tracheo-bronchial

lymph nodes, and the patient died of this condition six weeks after the operation.

Upon opening the chest at autopsy the most striking features noticed were the large and voluminous right lung, filling the right side of the chest and extending nearly over to the midclavicular line on the left side, and the small size of the left chest caused by the retraction of the walls on that side. Closer examination of the right lung showed compensatory hypertrophy and numerous areas of healed tuberculosis.

The left lung was collapsed below and tightly pressed against the mediastinum, a mass of thick walled bronchi and blood vessels, a small group of calcified lesions, and a very small amount of intervening compressed lung tissue, the whole adherent to the diaphragm and chest wall. The upper portion was more expanded and contained three or four cavities, filled with inspissated caseous matter, and surrounded by firmly compressed tissue containing bronchi, blood vessels and partially calcified fibrocaseous lesions.

I am indebted to Doctor L. U. Gardner of the Saranac Laboratory for the following report of the microscopic examination of this lung:—

"The tissue is chiefly composed of compressed alveolar walls, which have apparently undergone little or no change. Such tissue has obviously not been involved in the tuberculous process. Other foci undoubtedly once the site of active tuberculous lesions are now replaced by dense fibrous tissue, which shows the remains of air spaces as irregular, often elongated slits, lined by cuboidal epithelial cells. The original order of such air spaces is not now apparent. All the normal connective tissue, such as the septa, the pleura, the perivascular and peribronchial sheaths have been transformed into dense masses of fibrosis. The tuberculosis is limited to the large cavities noted in the gross, and to smaller calcified nodules. The cavities are lined by dense hyaline fibrous tissue, which contains a few small vessels, and is heavily infiltrated with lymphocytes. The inner wall of the cavities is generally smooth and shows no inflammatory exudate. The lumen contains a necrotic debris."

BRONCHIECTATIC LUNG ABSCESS

Operation—Recovery.

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According to the statements of Virchow, Biermer and others, bronchiectasis is, on the whole, a rare condition (four per cent of all post mortems). So also in his clinic Sokolowski finds bronchiectasis, especially with distinctly marked symptoms, relatively seldom. In his hospital station with a yearly number of 400 to 500 patients, he sees it barely a few times. In the practice of one of us, which is restricted to pulmonary tuberculosis, it occurs not more frequently than once or twice a year. Some years one or two cases will come under observation—the next year, perhaps no case. It is thus seen that the condition is infrequent.

We find bronchiectasis rarely in children. It is more frequent in young persons, also in those of more mature age, and in the aged. Many authors think that bronchiectasis occurs most frequently in old age. Sokolowski and Biermer are of the opinion that this view is incorrect. The former found the majority of his patients to be either young or middle aged. This is also our experience. Sex has no distinct influence, although many authors observed bronchiectasis more frequently in men.

The non-specific, sub-acute and chronic lung suppuration may be conveniently classified as (1) typical bronchiectasis, which usually is congenital; (2) the so-called bronchiectatic lung abscess; (3) the ordinary lung abscess, mostly consecutive to pneumonia and influenza.

One of the principal causes for the development of a bronchietatic lung abscess is the aspiration of a foreign body or foreign substance into the trachea and its divisions, particularly of blood,

mucus and infectionus material from the crypts of the tonsils during tonsillectomy and of stomach contents during general anaesthesia.

A malignant neoplasm of the lung proper, also a benign intrabronchial tumor can be the direct cause of a bronchiectatic affection of the lung.

With reference to therapeutics purely medical treatment can, of course, be put palliative; it cannot be successful as far as real improvement or cure is concerned. In one of our cases—a young girl of seventeen years—the sputum reduced spontaneously from 85 c.c. to 20 c.c. per day, and remained at 20 c.c. With the exception of insignificant dilations in children, or in otherwise very sound individuals, which may heal or cause no serious disturbances, the process always progresses towards deterioration of the affected lobe (or lobes) of the lung in the majority of persons.

Inasmuch as the disease much more frequently slowly progresses from bad to worse, the physician should always consider his patient with a view to possible surgical interference. He should take it for granted that with far advanced wide dilatations the disease will in time usually terminate fatally. He should therefore carefully consider whether it is not possible by other means, particularly by surgery, to obtain better results than by internal medication. The surgeon obtains successful results in other deep affections of the lungs and pleura, as unilocular circumscribed abscesses, localized gangrene and purulent pleuritic exudates; why should he not obtain successful results by operation on the bronchiectatic pulmonary abscess, and on bronchiectasis? We therefore wish to emphasize the point that every case of bronchiectasis should be carefully considered with a possible view to operation.

To mention just a few collective reports of older authors from the literature:—

The Cracow surgeon, Trzebieky, in his statistics of pneumotomy, published in 1892, which comprises 148 cases, gives 45 cases of surgical treatment of bronchiectasis, with not exactly brilliant results. Dr. Kijewski, in 1897, made a critical examination of the statistics of Fabrykant, and isolated cases of other surgeons, and added some descriptions of lung abscesses treated by operation from the clinic of Professor Kosinski. This also includes a case of bron-

chiectasis which was operated upon by Dr. Bukowski. This case afterwards progressed unfavorably. Hoffman collected twenty-three cases from the literature in which surgical treatment was carried out; twelve patients died, and in eleven cure or at least significant improvement is indicated. Toward the end of the last century Lenhartz, Quincke, Garré, Koerte, Tuffier, Brauer, and within the last two decades Sauerbruch, Meyer, Robinson, Lilienthal, Davies, Hedblom and others, also several prominent radiologists and bronchoscopists, have made important contributions to a better understanding of the disease and its treatment.

As just mentioned, the results of operation in bronchiectasis and bronchiectatic lung abscess formerly have not been favorable. Surgeons, therefore, correctly emphasized the fact that this disease in comparison with other purulent processes in the lungs, which are treated surgically, shows, relatively, the worst results. Kijewski, who discusses the question critically, asserts that even advanced large sized typical bronchiectases, so long as they do not produce any threatening disturbances, do not necessitate to open the chest. He emphasizes the fact, that if we open only one focus, by incision, we naturally make drainage easier from this one cavity only, without curing the other places from this one cavity only, without curing the other places from which the secretion is discharged.

However, if the contents of the dilated bronchi cannot be easily discharged and expectorated, on account of inflammatory or cicatricial strictures at the entrance into the cavities, if symptoms appear which point to incrustation of the walls, with retenion of the purulent secretion and septic absorption (continued, irregular fever) gradual exhaustion of the patient and eventual death are imminent. The not infrequent occurrence of acute meningitis and of brain abscess must also be considered.

In such a situation the surgeon should attack the disease which threatens the system, notwithstanding the fact that an operation less radical than extirpation brings perhaps no complete cure.

Limitation of the indication for operation solely to these cases will show much better results with the perfected technique of today than the older statistics which have been collected up to the present time.

In the course of years results have certainly much improved; they depend upon the type of procedure chosen, as well as the extent of the disease. Patients in the incipient stage of the disease and with changes in the lungs that are not extensive may improve a great deal, if aided by regular bronchoscopic aspiration. Under fortunate circumstances they can be cured by this means.

Patients with circumscribed bronchiectasis, so-called bronchiectatic lung abscess, who are treated with incision (pneumotomy) and prolonged drainage, have a good chance to recover from the operation, though in a more roundabout way, and may hope to be able to return to active life, even if not cured in the full sense of the word.

Resection of the affected lobe or lobes of the lung, is of course, the ideal operation. But even in carefully selected cases, and in the hands of the most experienced, it still has almost 50 per cent mortality. However, if successfully operated upon, such a patient will be completely cured.

In some cases of accurately circumscribed multiple cavity formation we may obtain, as just mentioned, real great improvement, exceptionally even complete cure of the disease by incision and prolonged drainage, carried out with the intention to produce a temporary or permanent lung fistula. The case to be reported illustrates in a satisfactory way the method which has been gradually and methodically worked out by one of us.

The fact that it is this circumscribed bronchiectasis (bronchiectatic lung abscess) which is the most suitable for operation, and that descriptions of successful results of the same are not very numerous in medical literature as yet, has urged us to publish this case.

The patient, a white man, was an engraver of forty years of age. His illness began in June, 1916, immediately after an operation for circumcision. Ether was used for general anaesthesis. High fever up to 104° F. set in soon after the operation and lasted for four weeks. One week after the operation he began to cough, and he then for the first time felt a sharp pain in the right axilla. The pain lasted one week. In June, 1916, he had night sweats and a right-sided dry pleurisy. He went to the Adirondack Mountains in August, 1916, and gained in weight and worked a little at

light work. Kept well until September, 1917. Temperature then began to be noticed, and a foul odor to the sputum. The temperature would run normal for some days, and then would rise to 101°-102° F. for several days. His past history showed nothing of importance. He had measles in childhood and whooping-cough at thirty-five years of age. He also had malaria. He is subject to colds in the head. Dyspepsia has been present since the onset of his trouble. He used tobacco rather too freely, but did not use alcohol. His health had always been good, though he was subject to tonsillitis and quinsy.

His wife died of pulmonary tuberculosis fourteen years previously. His family history was otherwise negative for tuberculosis.

His condition was that of a fairly well nourished white man; height, 5 ft. 11 inches; weight, 139½ pounds; normal weight, 160 pounds; appetite good; bowels regular; no evidence of indigestion; no sweats. His nose, pharynx and larynx were normal. The heart and abdomen were normal. The post cervical and axillary glands were slightly enlarged. There was marked clubbing of the fingernails. The pulse range was from 60 to 80. The temperature was unusually normal, except occasionally to 101° F. for one or two days. The urine was acid, and had a faint trace of albumin. There was no sugar. The sediment showed calcium oxalate crystals, a few pus and red blood cells; no casts.

He coughed and expectorated almost constantly, and the cough was always accompanied by expectoration. The expectoration had a very offensive, rather sweetish smell, and looked like pure pus. It varied in amount from sixteen to twenty-four ounces in twenty-four hours. It did not contain any elastic fibers, and repeated examinations failed to show tubercle bacilli. Cultures made from the sputum showed only pneumococci—no other organism. (The type of the pneumococcus was not determined). The sputum was inoculated into guinea pigs, and at the end of eleven weeks the pigs were killed, and no evidence of tuberculosis was found in them. Before the pigs were killed they were tested with an intracutaneous test of tuberculin, and failed to react.

The sputum came away from him very profusely when the head and body were held downwards, and on one occasion within fifteen

minutes he raised almost a pint of pus and some blood tinged sputum. Although he had almost constant cough and expectoration, the expectoration would come away very profusely within a very short time in the morning, and again later in the day.

The physical examination showed the following:—

Right Lung—Prominence of the clavicle; depression and deficient expansion of the right chest; some fine rales to the third rib; at the back there were some indefinite fine rales over the lower chest.

The left lung was normal, front and back.

The x-ray examination showed the following:—

Right Lung—Marked cloudiness of a patchy character throughout the upper three-fourths of the lung. These changes were more marked in the middle third, mainly inside the midsternal line. Some string-like shadows were seen throughout this cloudiness. The diaphragm was peaked.

The left lung showed two or three dense nodular shadows in the lower third.

The opinion on interpretation of the x-ray findings to the physician:—

Right Lung—Infiltration involving the upper three-fourths of the lung. These changes are atypical of tuberculosis as there is no real definite tubercle formation to be seen. The condition presenting these shadows is probably one of the following, in the order named: Lung abscess, gangrene, bronchiectasis. There is probably some pleurisy present on this side, with diaphragmatic adhesions. Another point confirmatory of this being a non-tuberculous condition is the freedom from any changes in the left lung.

Left Lung—Probably one or two calcified tubercles at the base.

X-ray examination of the cranial sinuses showed a fairly marked clouding of the right antrum.

On two separate occasions attempts were made to compress the right lung by nitrogen gas, but no gas could be introduced.

Operation was performed early in January, 1918, under regional and local anaesthesia, and was done without causing the patient any appreciable pain. The upper lobe of the lung was found attached to the costal pleura, and the abscess was struck two and one-half

inches below the surface within the upper lobe and severe hemorrhage being encountered. He stood the operation nicely.

Details of Operation.

Incision slightly convex downward in second intercostal space, division of major and minor pectoral muscle, bluntly, in the direction of their fibres with double ligation and division of crossing blood vessels; retraction of the muscles with blunt hooks (this act seems to be slightly painful); the muscles obstruct field, therefore division of major pectoral downward and of minor upward at right angles to direction of fibres for some distance; now access easier; resection of four inches of second rib; careful division of intercostal muscles proves lung not moving, pleural leaves evidently adherent; aspiration with long needle of large caliber in bed of resected second rib, needle being pushed forward in straight direction backward, with clear sensation of penetrating infiltrated tissue and then suddenly entering cavity. Aspiration draws some arterial blood with distinct pus-flakes in greater quantity; incision of lung with cautery, carefully following needle as guide; on reaching latter's tip the cavity is struck and widely opened; it contains coagulated masses of fibrin of various size and shape, and of the same foul odor as the sputum has shown for many months. The finger, introduced, palpates an irregular cavity with a perpendicularly forward rising, resistant arch, evidently the wall of a distended larger bronchus; upward the flexed tip of the finger enters a smaller pocket "around the corner." The cavity lies fully two and one-half inches below the surface of the lung. Drainage with split rubber tube, iodoform and sterile gauze tampons; loose dressing. Patient in splendid condition after operation. One week later first change of dressing. Lung wound now represents large cylindrical hole, in depth of which one large and several smaller opened bronchi are visible. Retamponade. In course of further after-treatment care is taken to establish a skin lip-fistula; after its definite formation the rubber drain is removed.

In January, 1919, the patient reported that he was alive and in fine trim; that he coughed only a few times in twenty-four hours, that he had hardly any expectoration, and that he was able to work.

In July, 1919, i.e., nineteen months after operation he reported for examination at Saranac Lake. His pulse was 60 to the minute and weight 157 pounds. He had been at work for one year, and was a chauffeur for the past three months. He had slight morning cough and expectoration. He felt in perfect health. There was no tubercle bacilli in his sputum. There was a scar about six to seven inches long downwards and forwards over the front of the right chest. The examination of the chest was as follows:—

Right Lung—Intense blowing breathing over a fistulous opening situated in the front of the chest. Air escapes from the opening. Back—moderately fine, faint rales to the eighth dorsal spine.

Left Lung—Impaired resonance to second rib; no rales. Back —moderately fine, faint rales over the base.

X-ray examination, July 22, 1919, nineteen months ofter operation:—

Right Lung—Anterior portion of the second rib resected. As regards the intra-pulmonary changes, there are many fibrous strings and striations scattered throughout the lung (upper half or two-thirds). The previous homogeneous cloudiness seen in this region has disappeared almost completely. However, there is some cloudiness, localized in places, in the descending trunk region. It is difficult to say positively just what is causing this haziness. There is still slight evidence of diaphragmatic pleurisy.

Left Lung—There are no definite changes to be made out on this side. However, there is a little more cloudiness around the root region and extending into the lower half. This, however, is not definite enough to interpret.

Today, four years after operation, the artificial pulmonary fistula is still present; its skin entrance is collapsed; on separating its lumen and with the patient's lips closed and nostrils compressed, the required quantity of air for ordinary respiration can be easily drawn through the fistula. There is no cough, no expectoration, no foetor. Patient is at work, in splendid physical condition. He is opposed to having the fistula closed at present.

NON-TUBERCULAR COMPLICATIONS OF TUBERCULOSIS.

By R. H. M. LANDIS, M.D.,

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In considering the non-tuberculous complications of tuberculosis I shall make no attempt to cover the entire list, but confine myself to those which, in my experience, have been encountered most often. There are many traditions handed down to us in regard to the rarity or the frequency with which certain conditions occur in tuberculosis. For example, it has been long believed and is still believed that a gouty individual rarely becomes tuberculous; on the other hand it is known that diabetics are quite apt to become so. The truth of the matter is that, with few exceptions, there is scarcely a disease, whether acute or chronic, that may not develop in an individual who has either active or latent tuberculosis. Some diseases occur so infrequently as to be viewed in the light of rarities; others are combined with tuberculosis so frequently that in any considerable number of cases of the latter disease they will be encountered quite often.

It is essential that those who handle tuberculous patients should have a knowledge of these more common complications. In the first place, methods of treatment often must be varied to meet the particular individual. In the second place, a latent and unsuspected infection may retard improvement and may be the actual cause of a persistent fever, for example, which is wrongly attributed to the tuberculous lesion. Those of us who are constantly seeing tuberculosis must always be alert to the possibility that there may be other contributing factors and this is especially true in those cases in which the symptoms remain stationery or undergo exacerbations from time to time.

Diabetes.

It has long been recognized that diabetes and tuberculosis occur relatively frequently in the same individual. It is a difficult combination to deal with. Prior to the introduction of the modern

method of handling diabetes the usual procedure was to ignore the diabetes and with perhaps a few minor restriction as to diet, follow the course usually prescribed for tuberculous individuals. The results were almost universally bad. In dealing with this complication the first requisite is to control the diabetes. Unless this is done little or no progress can be expected.

The management of tuberculosis under these circumstances presents an interesting problem. The treatment of tuberculosis rests on three basic principles:—rest, a generous diet, and an abundance of fresh air.

In diabetes, on the other hand, the patient is either starved for a brief period or placed on a restricted diet. Under the best of conditions, the total amount of food one can give these patients is far below that usually thought necessary for a tuberculous individual. Furthermore, a moderate amount of exercise is desirable for the uncomplicated diabetic.

It can be readily seen that in dealing with this combination of diseases we must put aside one of the three main objectives aimed at in the uncomplicated cases of tuberculosis, namely, a gain in weight. Instead of gaining these patients will inevitably lose weight, sometimes alarmingly so. Nor can this loss, as a rule be entirely restored owing to the relatively low intake necessary to keep them sugar free. As a compensation for the loss of weight the patient improves in other respects, especially as regards toxic symptoms (fever, tachycardia, sweating, etc.)

While my experience up to the present time is limited, directly or indirectly, to about thirty cases, I am still convinced that the control of the tuberculosis in such cases is absolutely dependent on getting the patient sugar free and keeping him so. It is, of course, to be borne in mind that either one of the diseases may be so severe as to preclude recovery even if existing alone. But even in the inevitably fatal cases they can be made more comfortable if the diabetes is first controlled.

Pregnancy.

There is no phase of the tuberculosis problem that is more important to the general practitioner than that of pregnancy as the incidence of the pathological and the physiological condition is a

frequent one. In 1913 Bacon estimated that 32,000 tuberculous women became pregnant annually in the United States and added that between 44,000 and 48,000 women of the child-bearing age die of tuberculosis every year. There is no doubt, in my opinion, that pregnancy does exert a distinct influence on the occurrence of tuberculosis in women. Several years ago I studied a number of women at the White Haven Sanatorium and found that over twenty per cent of those who had borne children dated the onset of their disease from a pregnancy.

A latent tuberculosis may be activated or an arrested case may relapse as the result of pregnancy under the following circumstances: In one group the tuberculosis may manifest itself during the pregnancy, often within the first few months. In the other group the woman remains well during her entire term but shortly after confinement develops pulmonary symptoms, often extremely acute in character.

Why the symptoms develop during pregnancy is not clear. In some instances it would seem to be associated with the exhaustion and lack of nourishment due to severe vomiting but in others is no such factor. In those who manifest evidences of the disease following confinement, the violent exertion that characterizes many labors is commonly accepted as the probable cause of arousing to activity a latent lesion.

If one reviews the literature on this subject, the outlook is not a promising one. This is ascribed to the fact that the majority of pregnant women are delivered either by the general practitioner or by the midwife, and only a relatively small portion are seen by the specialist. It naturally follows that the specialist is called in consultation in the worst cases, and that the worst cases are likely to be sent to hospitals for delivery, where again they fall into the hands of the obstetrician. Thus the obstetrician is especially prone to see those patients who are doing badly, and this is doubtless a factor in the high mortality and morbidity as usually reported in this condition.

The results obtained in the Prenatal Clinic at the Phipps Institute presents an entirely different picture from that usually painted. In a report made by C. C. Norris and myself five years

ago, we dealt with eighty-five cases. These cases consisted of about the ordinary run of pulmonary tuberculosis and were not of the class usually seen by the obstetrician.

Of the 85 cases, 69 or 81.1% showed no change as the result of their pregnancy; two patients became worse and six died. In three cases the pregnancy was interrupted as the result of the pulmonary condition.

In dealing with this combination one has the choice of three different opinions: (1) That the uterus be emptied in all cases regardless of whether the patient presents symptoms of active tuberculosis or not; (2) That under no circumstances shall the pregnancy be interfered with; (3) That one shall pursue a waiting course. If there are no symptoms allow the pregnancy to continue. If, however, the women develop symptoms which persist and become worse a therapeutic abortion should be done, providing the pregnancy has not advanced beyond four months. If not seen until after the fourth month, interference is not advisable as emptying the uterus in the later months of the pregnancy has little favorable effect on the pulmonary lesion.

The last named course is the one we have adopted at the Phipps Institute and as already mentioned, active interference has been deemed necessary in but three of eighty-five cases. It is fair to state, however, that this number would probably have been greater had some of the women been seen prior to the fifth month of the pregnancy.

Nephritis.

Some evidence of chronic inflammatory changes in the kidneys is present in the great majority of cases of pulmonary tuberculosis coming to autopsy. During the terminal minutes of the disease traces of albumen and the presence of a few hyaline or finely granulated casts are commonly present in the urine but it is rare, in my experience, to meet with severe grades of nephritis in tuberculous individuals. I cannot recall more than two cases in which the kidney function was seriously disturbed. In both these cases there were large amounts of albumen, numerous tube casts, a scanty output of urine and marked edema. In addition functional tests showed

an extreme grade of retention. The dietary restrictions in these cases make them even more difficult to handle than diabetics.

Heart Disease.

Narly three-quarters of a century ago Rokitansky enunciated the doctrine that venosity of the blood was antagonistic to the development of pulmonary tuberculosis. Among the conditions producing venosity he included all forms of heart disease, both acquired and congenital. Since his time there have been many observers who considered that left-sided heart lesions, especially mitral stenosis, did exert an inhibitory acting on the growth of the tubercle bacillus. A number of studies have been made to prove or disprove this fact. Norris (G. W.) after a full review of the literature and study of a large number of protocals concluded that heart diseases exerts little if any influence on the pulmonary lesion either curative or inhibitory. C. Y. White, in a study of autopsies on tuberculous individuals dying at the Phipps Institute found that the valve leaflets were functionally disabled in six per cent of cases. In his opinion passive congestion of the lungs does not exert any effect on the development of tuberculosis.

In discussing the relationship between cardiac diseases and pulmonary tuberculosis there are three types of cases to be considered: (1) pure cardiac cases; (2) those in which there is an obvious cardiac lesion and possibly a latent pulmonary infection; and (3) those in which cardiac disease and tuberculosis are undeniably present.

It is well known that mitral stenosis especially is apt to be mistaken for tuberculosis because of the cough, blood, spitting, often loss of weight and the presence of rales. As a rule it is not difficult to determine that the cardiac lesion is the real cause of the trouble. In a certain number of instances physical signs may point to the possibility of an associated tuberculous lesion. Sputum examinations and consultation with a roentgenologist will determine whether one has to deal with a complicated or uncomplicated case.

Within the past year I have seen two cases of mitral stenosis with fibrillating auricles and with tubercle bacilli in the sputum. My own experience is that individuals with enlarged hearts, definite

valvular lesions and pulmonary tuberculosis are encountered relatively frequently and that there is little to warrant the view that cardiac disease is antagonistic to tuberculosis.

When the two conditions occur together the treatment of the tuberculosis is that of the uncomplicated case although occasionally one is encountered that stands, even moderate altitudes, very badly. If the compensation shows signs of breaking one should proceed also as though the case were uncomplicated.

Syphilis.

It is inevitable in view of the frequent incidence of both syphilis and tuberculosis that these two diseases will be encountered often in the same individual. It has long been recognized that when both conditions are active in the same person the combination is a vicious one. Distressing examples of this are sometimes seen in young prostitutes. When tuberculosis develops in an individual with an old and long standing luetic infection the tuberculosis does not differ essentially from that occurring in an individual free from syphilis. This fact should be kept in mind, however. Many, if not most of these individuals should receive anti-syphilis treatment. In not a few of them the syphilis although latent and presenting none of the common stigmata, may at the same time exercise a retarding effect on improvement just as a chronic focal infection.

The frequency with which tuberculosis and latent syphilis exists in patients coming to the Phipps Institute has made it a necessity for us to establish a special treatment clinic for this group. This necessity has arisen largely because of a very large Negro clinic and as is well known a dual infection in members of this race is very common.

Our procedure is as follows: If the patient is tuberculous the initial dose of arsphenamine is .3 of a gram. If this produces no untoward effects the subsequent doses are .6 of a gram. Six injections are given and then the patient is placed on some form of mercurial medication for six weeks. If the Wassermann reaction remains positive both courses are repeated. Many of these chronic luetic cases are extremely difficult to get Wassermann free. If after repeating the treatment the reaction remains positive they are

advised to repeat the single course of treatments once a year.

Iodide of potassium is never used in luetic individuals who in addition are tuberculous. In my judgment the use of this drug in the presence of a tuberculous lesion is dangerous. I have seen latent arrested cases of tuberculosis aroused into activity by the administration of small doses of iodide of potassium for diagnostic purposes.

Pneumonia.

Pneumonia in its relationship to pulmonary tuberculosis presents two problems: (1) That of distinguishing between true lobar and tuberculous pneumonia and (2) that of determining whether in a tuberculous individual an acute febrile attack associated with signs of consolidation in previously healthy lung tissue is the result of a lobar pneumonia or a rapid extension of the tuberculous process.

It is not sufficiently appreciated that pulmonary tuberculosis often first manifests itself as an acute infection. There may be a chill or chilly sensation, a rapid rise in the temperature and a corresponding quickening of both the pulse and respiration rates. In addition there may be blood-streaked sputum. Associated with these symptoms are signs of partial or complete consolidation in one lung and, this should be emphasized, it is an upper lobe involvement. Such a case may terminate in several ways: (1) It may progress rapidly and terminate in a few months or even weeks as an acute tuberculous pneumonia; (2) the acute symptoms may subside and the temperature fall to normal in two or three weeks; (3) it may shift into the ordinary chronic ulcerative type of case.

It is noteworthy that many of these cases present every feature of a lobar pneumonia and for a week or so no suspicion of their true nature occurs. In not a few instances, when it finally becomes apparent that tuberculosis is present, it is said that the latter was a result of pneumonia.

When lobar pneumonia develops as a complication in cases of pulmonary tuberculosis it is often impossible at the onset of the trouble to determine whether the acute disturbance is a true pneumonia or an extension of the tuberculous process. The following cases will serve as illustrations. A physician with a well marked, but arrested lesion involving the greater part of his right

upper lobe was suddenly seized with chilly sensations, vomiting, marked prostration and a rapid rise of temperature. He had previously been in excellent health although his sputum always showed the presence of tubercle bacilli. Examination of his chest a few days later revealed a consolidation of his right lower lobe. When I saw him, he had been ill twelve days and the temperature was continuous, varying from 101° to 103° F. He was fearful that he had suffered a relapse. In this case there was nothing to guide one as to whether it was a lobar pneumonia or sudden extension of his tuberculosis. A day later he had a crisis and the lung rapidly cleared up.

Another somewhat remarkable case was that of a woman with moderately advanced lesions in both upper lobes. Her temperature had been normal for two months and she was taking two hours exercise daily. She was seized suddenly with a chill, marked prostration and a high fever. A few days later involvement of the entire left lower lobe became apparent. She ran a febrile course of ten days when her temperature reached normal by lysis. Recovery was apparently complete. Ten days later the same phenomena recurred with involvement of the right lower lobe. This attack subsided as the first had done. These cases differ from the first mentioned group in that the presence of tubercle bacilli offer no aid as they are already present. X-rays are helpful at times but unfortunately one is often unable to obtain this assistance.

It is interesting to note that even when the lung which is already tuberculous becomes pneumonic, no activity of the tuberculous lesion occurs. This is surprising in view of the fact that ordinary "colds" are so often the means of bringing about relapse. In this connection it might be noted that typhoid fever, which is so commonly complicated with a bronchitis, is usually well borne by tuberculous patients.

Malignant Disease.

The association of malignant disease of the lungs and tuberculosis is unusual. Indeed, one may go farther and state that a tuberculosis individual only infrequently develops malignant disease in any part of his body. Until a few years ago my own experience indicated that the co-existence of these two conditions was a rarity

but I have since had three patients with advanced pulmonary lesions who developed a malignant tumor elsewhere in the body. Among sixty cases of malignant disease studied by Ross there were two cases in which tubercle bacilli were found in the sputum. He notes that the frequency of healed tuberculous lesions found at the autopsy is of no significance as they are found with equal frequency in individuals dying of other diseases.

Focal Infections.

Focal infections offer two problems. First as to whether a complex of indefinite symptoms is tuberculous in origin. For example, a patient may complain of lassitude, have a slight but persistent fever, loss of weight and in certain latent sinus infection also have considerable cough. Such cases are often mistaken for tuberculosis and treated as such for some time. It is only when failure to obtain improvement occurs that the suspicion arises that the source of the trouble may be another type of infection. Secondly, a definitely tuberculous individual may fail to improve or may develop what seems to be slight exacerbations of the disease when the trouble is in reality due to an associated focal infection. One should always be alert for such possibilities in cases which fail to improve and for which there is no satisfactory explanation. The trouble may be in the tonsils, carious teeth, etc. I have had several humiliating experiences in this regard. A man under my care had improved satisfactorily up to a certain point. Then for a period of nearly six months he was subject to recurring attacks of fever, associated with lassitude and some loss of weight. I persisted in ascribing these attacks to his pulmonary lesions. Finally he was given a general overhauling, and a chronic sinus infection was found. This was drained and he speedily went on to a complete recovery. A chronically diseased tonsil in another case so retarded recovery that nothing was accomplished until the offender was removed; but it took me three months to come to this decision.

Neurasthemia.

As in the case of nearly all the complications mentioned neuras-

themia presents the problem of differential diagnosis as well as its recognition as a complication.

For some years I have recognized a type of tuberculosis in which the outstanding feature is the combination of symptoms embraced under the term neurasthemia. Such patients often suffer for years from some ill defined malady; they tire easily, especially at the end of the day, and are extremely susceptible to fatigue on the slightest exertion. The blood pressure is low or is lowered as the result of exertion. Respiratory symptoms are slight or entirely absent. Recently this type has been described under the term of masked or occult tuberculosis.

Not infrequently patients with definite clinical tuberculosis suffer in addition from marked nervous symptoms of a neurasthemic nature. In many the nervous manifestations overshadow the pulmonary symptoms. Many such patients are best handled by the neurologist. They require seclusion, prolonged rest and the general routine known as the Weir Mitchell Rest Cure. Indeed in Mitchell's original exposition of the rest cure many of his cases were undoubtedly of the masked type, or those frankly tuberculous with marked nervous symptoms.

Finally neurasthemia becames a problem in many arrested cases. This is notably the case in those in whom the rest treatment has been unduly prolonged. Not infrequently such patients become unduly self-centered, timid and unable to do anything. They complain of fatigue after the slightest exertion and in addition become the victims of all sorts of vague symptoms. In not a few of them it can be said, that while their tuberculosis has been arrested, their second state is worse than their first as they become a burden and annoyance to themselves and every one about them. It often takes drastic measures to restore their self confidence and divert them back into normal channels.

CLINICAL OBSERVATIONS ON THE MANAGEMENT OF DIABETES MELLITUS.

By LAWRENCE LITCHFIELD, M.D.,

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Although there are references to diabetes in early Egyptian, Arabian, Hindu, and Galenic Greek writings, the diabetic problem may be said to have been really broached in England about the middle of the seventeenth century by Thos. Willis of "Circle of Willis" fame, taken to France by Claude Bernard and his pupils about the middle of the last century, appropriated by Germany, von Jaksch, Naunyn, Minskowski, and their contemporaries, and finally transplanted to the United States, apparently for final settlement, about the beginning of this century. Atwater, Folin, Lusk, Allen, Joslin, Macleod, Woodyatt, Newburgh, and others have done much to bring about this consummation.

The treatment of the diabetic patient, however, is not the same thing as the study of diabetes. The latter is pursued by group methods, logarithms and the slide rule. The former is mastered by the meticulous attention to individual details: in the one we seek laws, while in the other we study the exceptions. The clinician may be said to study the problems from the point of view of the patient himself. John Doe is not merely a case of diabetes, but a man of a certain definite weight and a certain definite height, coming from a particular family, handicapped by definite habits and heredity, and with a very complicated disturbance of his metabolism.

In discussions on the management of diabetes some confusion has arisen from a lack of precise definition as to the type of cases that are being discussed. The very severe cases present an entirely different problem from the moderate cases, both as to methods of treatment and as regards the objects or results to be aimed at. The majority of recent discussions have concerned themselves for the most part with the management of desperate cases in which the most that can be expected is to keep the patients alive and, if possible, in

tolerable comfort, for a more or less limited period. On the other hand there is much to be said as to the management of the milder cases in which proper treatment may lead to the absence of disease and to efficient, happy, self-supporting lives of unlimited term. It is in regard to these cases that I wish to present to you certain clinical observations.

Fat Metabolism.

I have seen an increase of 10 gm. of fat to a greatly undernourished child of three years who was getting less than 10 gm. of fat daily, followed by the excretion of 9 gm. of keton bodies in the urine within twenty-four hours with concomitant signs and symptoms of mild acidosis. I have seen two pregnant women develop signs and symptoms of severe acidosis after the sudden elimination of fat from their diet during a gradual reduction of the diet on account of glycosuria. I have seen apparently similar cases, but not pregnant, bear greater and more rapid reduction of the diet without any evidence of acidosis. I have seen a moderate increase of fat in the dietary followed by an apparent reduction of the carbohydrate tolerance. I have seen a patient who was getting only 500 gm. of 5% vegetable and half a grapefruit, with no other nourishment, stand the sudden addition of 90 gm. of fat, and 24 gm. of protein, without the appearance of glycosuria or acidosis. I have seen moderate additions of fat to the diet followed by evidences of acidosis. I have repeatedly seen patients who had been getting along comfortably with untreated diabetes, develop acidosis and go into coma when suddenly put on the old carbohydrate free diet, although the 10% of the fat and 58% of the protein in the diet which they took should have yielded enough carbohydrates in their metabolism, according to certain alleged laws, to have taken care of the fats given. I have seen other patients under the old régime who stood the radical change of diet just mentioned without accident, but with apparent benefit.

It is my impression that pregnant women are in much graver danger of acidosis from endogenous fat metabolism than other diabetic patients. I have seen a fat woman show a negative ferric chloride test for several days while she took no exogenous fat and

develop positive ferric chloride reaction after a couple of days with a ration of 90 to 100 gm. of fat daily.

These desultory observations on fat metabolism satisfy me that I can not predict a'priori how any individual patient will behave as regards endogenous or exogenous fat metabolism,—that is when thrown upon her own reserves for fuel, or when fed fat freely.

When faced with alarming symptoms of acidosis, I follow with great satisfaction the methods developed by Joslin with the addition, when coma threatens, of the intravenous use of 5% dextrose solution,—200 to 500 cc.—with 500 cc. normal saline or Ringer's solution.

The importance of the consideration of the patient's weight in the *management* of diabetes, as well as in the *avoidance* of diabetes, can not be overestimated. Many fat diabetics may need no other advice than to bring their weights down, and keep them within normal limits on a properly balanced diet.

If my diabetic patient is overweight, I allow him to burn his body fat under careful surveillance,—daily ferric chloride tests in certain cases,—and at the same time work out his carbohydrate tolerance, and try to kep him in nitrogen equilibrium. In arranging reducing diets for these fat diabetics it must be borne in mind that most of these patients have been eaters of bulky diets, and if this habit is suddenly changed by putting them upon a diet of small bulk, serious constipation and possible autointoxication will result. Therefore, 5 and 10% vegetables, and sometimes partially desugared canned fruits should be prescribed in sufficient amount to give their reducing diet about the same bulk as the diet they previously enjoyed. Paraffin oil, or agar may be used is necessary.

If my diabetic patient is underweight, I give him fat to his point of tolerance, or to a satisfactory daily caloric intake. It is in some of these cases that we may be fortunate enough, by carefully training and modifying the habits of the organism, to change the individual into an Eskimo, as Joslin so happily expressed it. While I believe in regulating the dietary by blood sugar determinations, when practicable, at the same time, if blood can not be obtained easily for this purpose, I feel that one may proceed safely by keeping the urine constantly sugar-free and watching closely the general

appearance and the sensations of well being of our patients. A smile, or a sparkling eye, does not go with impending trouble.

Diabetes and Obesity.

The intricate relation of diabetes mellitus to obesity has not been entirely worked out as yet. Both are conditions of disordered metabolism. Although they are so closely related, neither necessarily implies the other. I have recently seen a man weighing 343 pounds who is the only known case of glycosuria in a very large family with a most remarkable record for health, vigor, and longevity, as well as for obesity.

In another family of six brothers and sisters, five weighed over 250 pounds, and of this five, four died of diabetes.

In this connection I wish to report and compare two fat diabetics. Both are men of three score years and both are leading very active lives. One is a practicing physician. The one has been under treatment two and one-half months and the other seven months. Both became sugar-free promptly and have remained so. Neither has shown any symptoms of acidosis and rarely a slight ferric chloride reaction. Both show albumen and occasionally a few hyalin and finely granular casts. Both show moderate hypertension, with moderate enlargement of the heart and good compensation. Electrocardiograms of both patients show left ventricular preponderance and rather low voltage. Dr. R. R. Snowden of Pittsburgh, has very kindly made the following laboratory studies of these cases:

	The one	The other
Basal Metabolism	+3	+4
Phthalein (subcutaneously) returned in two hours	50%	55%
Blood chemistry (non protein nitrog.)	40	46
Creatinnin	1.3	1.2
Uric Acid	2.85	2.75
Chlorides	640	607

They were given, each, 50 gm. of glucose in 250 cc. of water in the morning fasting. The one who came under treatment seven months ago, weighing 260 pounds (5′ 9″ tall), with a blood sugar of 420 mgm. per 100 cc. of blood, and who reduced fifty pounds in the first four months,—according to the method suggested above,—

who walks from four to seven miles daily with pleasure, developed no glycosuria, and starting with a blood sugar of 110. showed a perfectly normal curve,—160. in 30 minutes, 170. in 60 minutes, 130. in 90 minutes, 110. in two hours, and 90. in three hours. The other who began his treatment two and one-half months ago, weighing 215 pounds with a blood sugar of 130 mgm. and has been on an ordinary reducing diet, with a loss of only ten pounds developed glycosuria in thirty minutes which lasted for two days. His blood sugar started at 90. ran to 260. in 30 minutes, 270. in 60 and 90 minutes, 250. in two hours, and 240. in three hours, a so-called "diabetic curve."

I do not think it can be inferred, however, from these glycemic curves, that the former has been a simple case of obesity which has come to normal by the reduction of fifty pounds in his weight, or that the other is a true diabetic because of his ready development of glycosuria and the so-called "diabetic curve," after the glucose test. I believe the difference is due to the fact that the metabolism of the one patient has been gradually brought to normal, or nearly so, by a long course of dieting and exercise, and the reduction of his weight from 260 to 210 pounds, while the other has been under treatment but a short time and his weight has been reduced only from 215 to 205 pounds. It will be excedingly interesting to follow the effect of further intensive dieting and to get his glycemic curve when his weight has been reduced proportionately to the reduction in the other case. It is interesting to know that this man has complained much more of hunger than the man whose weight went down much more rapidly. This difference is probably a psychic one.

Sugar-Free Kefir.

Whether patients are to be started on relatively high fats, or brought up to high fats gradually, fats are bound to play an important part in the handling of many mild or moderately severe cases of diabetes, particularly the cases that can still lead active lives. Another class of cases, where a form of fat which can be well tolerated will prove to be a great asset, is the diabetic with a tendency to diarrhoea, with obstinately alkaline stools, and strongly alkaline urine. To meet both of these indications, I have had

developed a sugar-free kefir made for me by a Pittsburger, who has been very successful in making and marketing a kefir under the name of "lackzoom." It is made, he claims, of a pure culture of bacillus bulgaricus. I have used this product very successfully in a number of cases, but I have noted that the effect on the alkalinity of the stools was not prompt or constant, consequently I have been greatly intersted in the work reported by Rettger and Cheplin[1]. Inasmuch as bacillus bulgaricus will not flourish and multiply in the human intestines, as pointed out by the authors mentioned, I believe that it would be preferable to prepare my sugar-free kefir with bacillus acidophilus for diabetics with diarrhoea. Wheter or not the acidophilus kefir would have an advantage over the bulgaricus kefir in other diabetic cases,—that is cases without diarrhoea,—will have to be determined by further experiment. I am at present interested in determining whether or not a pure culture of bacillus acidophilus will exhaust the lactose in the kefir as well as the bacillus bulgaricus does, and if so, what modification of the carbohydrate ration will be necessary or advisable for the diabetic on account of the carbohydrate of the diet which will be used up by the activities of the bacillus acidophilus in the intestinal tract. The possible influence of the bacillary acid on the diabetic patient must also be investigated.

If the sugar-free kefir is standardized at a definite composition, as 3% protein and 10% fat, any desired dietetic formula can be obtained very readily, by the addition of varying amounts of water and washed cream. For example: One pint of kefir will yield, protein 15, fat 50, and the addition of 100 cc. of 40% washed cream and 400 cc. of distilled water will yield a liter containing, protein 15, fat 90, which is very palatable.

Emulsification.

Occasionally diabetic patients in whom fats are being pushed, will show a tendency to loose movements containing a considerable amount of undigested fat. In these cases I have found that the fat is usually assimilated if it is broken up in the form of an emulsion,—a carefully made mayonnaise being assimilated, when a French dressing is not, and whipped cream being taken care of much better than plain cream. I have found it practicable to have

the cream whipped and washed free of lactose at the same time, using ice water with lumps of ice in a cocktail shaker, or a mason jar, in the old familiar way. The whipped cream may be slightly sweetened with saccharine if desired.

Diabetic Gangrene.

I have seen a diabetic, well past middle age, develop gangrene in both feet, at different times, recovering each time spontaneously, with the loss of about a quarter of the foot and several toes, but with good functional use of the feet for years afterward, and with a subsequent severe attack of diabetes without any evidence of gangrene. It is generally accepted that these cases should be allowed to take care of themselves, if possible, without surgical interference, but if toxemia develops and progresses, and if lines of lymphangitis appear, extending up the leg, an immediate high amputation is the safest course, regardless of the Benedict or ferric chloride reactions. On account of the influence of septic and toxic processes on metabolism, and particularly on the carbohydrate tolerance, the source of an increasing toxemia must be removed before we can hope to cope with the diabetic condition.

We must not lose sight of the fact that imminent danger in diabetes is always from acidosis. I believe that at a critical time it is better to have a trace of sugar in the urine than to run the risk of not giving as much carbohydrate as the patient can metabolize, and, furthermore, I believe that a fairly heavy reaction to the ferric chloride test may be ignored if the urine is examined two or three times daily and the patient's general condition is steadily improving.

The following case presents so many points of special interest and importance that I will report it in detail:

Mrs. G. M., age 50, four children, one miscarriage, climacteric at 47. Much trouble with the teeth, the last of which were removed twenty years ago. Hay fever annually since early married life. Weight, on admission to the hospital, 200 pounds, height 5′ 9½″, maximum weight 255 pounds in 1913. Six or seven years ago noticed loss of weight, polyuria, polydipsia, and recurrent eczema, with burning and itching of the skin. In 1918 she had the 'flu and in 1921, or last December, she had what was called the "grippe."

After each of these infections her general condition seemed to be worse. Her family are all heavy, one of her sons weighting 270 pounds but no other cases of diabetes have been recognized in the connection. Shortly after an attack of grippe last December a dark spot appeared on the right great toe, beginning from a slight trauma, and soon after a crateriform ulcer began to develop on the ball of the same foot, from cutting a corn too close.

She was admitted to the hospital on the 21st of March, 1922, with dry gangrene of the right toe, a crateriform ulcer the size of a dime, and very painful, on the ball of the same foot, the third toe swollen and slightly cyanotic. No pulsation could be felt in the dorsalis pedis, or the posterior tibial arteries. There was some tenderness over the instep, slight tenderness in the right iliac region and along the border of the liver. The urine was 1020, a trace of albumen, a strong reaction for sugar, no diacetic acid, no casts. The blood Wassermann was negative with the plasma C 0.2 65 vol. per cent. Blood sugar 229 mgm. She was put on 150 gm. 5% vegetables, oatmeal porridge 60 gm., half a grapefruit, butter 10 gm., lackzoom or buttermilk two or three glasses, coffee and broth ad. lib. (This was an approximation of her diet on coming to the hospital standardized without radical change for observation and represented about 28 protein, 39 fat, 37 carbohydrate). Water was ordered in sufficient quantity to keep the specific gravity of the urine in the neighborhood of 1010. The carbohydrate was reduced gradually, and on the 27th of March the Benedict and ferric chloride tests were both negative. There was a trace of albumen in the urine but no casts. Her diet was gradually increased and her condition seemed to be improving from day to day, but on the 29th her blood sugar was reported as 233 mgm. and from that time she was starved with the purpose of reducing the blood sugar.

On the 3rd of April the blood sugar was 209 and on the 8th of April 176. Her blood pressure remained about constant, being recorded on the 3rd as 164/84. She bore the starving badly, lost her appetite, strength, and buoyancy, while her foot became progressively worse. It was very difficult to get blood for the blood sugar tests and the patient became more and more nervous and discour-

aged. The temperature and pulse showed a slight upward tendency and the patient was very evidently becoming toxic.

On the 11th of April the diet had been increased to 500 gm. of 5% vegetables and half a grapefruit, coffee, and broth, and abundant water supply. It was evident that with an increasing toxemia we could not expect an improvement of carbohydrate tolerance and the diet was increased radically to 600 cc. of sugar-free kefir, 150 cc. of 40% washed cream, and a quarter of one grapefruit. This represented, protein 29, fat 90, carbohydrate 6. Adding 10% of the fat and 58% of the protein for this diet gives a potential carbohydrate of 31 gm. At once the patient's general condition began to improve, but the temperature and pulse were still tending upward and two or three tender red lines of lymphangitis were first noticed on the 13th extending upward from the foot and by the 16th had crept up to the upper third of the leg. No pulsation could be felt in the popliteal region, which however contained considerable adipose tissue, but tenderness was elicited here in searching for the artery.

On the 16th the blood pressure was 166/86, the pulse had been 120 for three days, the temperature varying daily between 99 and 101°. The urine showed a positive Benedict and slight ferric chloride reaction. The patient's condition was evidently very grave. The limb was amputated between the middle and lower third of the thigh under light nitrous oxide and oxygen anaesthesia, proceded by two hypodermic injections of morphia, one-sixth of a grain each. Spinal anaesthesia, which for obvious reasons might have been thought desirable in this case, was decided against by Dr. R. R. Higgins,—whom we regard as an expert on this subject,—because this patient seemed to have a marked loss of elasticity in the arterial tree; she had been sick for a comparatively long time; was evidently quite toxic and gave us the impression of having a very low margin of resistance. All patients show a marked drop of their blood pressure, under spinal anaesthesia, and in cases like the present it is sometimes impossible to bring about a reaction from this depression, and the suggestion of local anaesthesia was discarded to avoid traumatising the flaps by the infiltration of the anaesthetic. The anaesthetic was maintained one hour without any cyanosis. The pulse grad-

ually rose to 140 during the anaesthetic, at the same time a vein was cut down upon and 500 cc. of 5% dextrose solution and 500 cc. of normal saline was given intravenously. This was given to counteract the general toxemia and as a safeguard against acidosis. The flaps were coapted carefully and adequately stitched. The patient required no anodyne on coming out of the anaesthetic and rested better than she had rested for several nights, voided urine freely, and took water abundantly during the night by mouth, and nourishment the next morning, the same as given immediately before the operation.

The third day from the operation there was nausea and vomiting and nothing was given by mouth for twelve hours, while enteroclysis of plain tap water was pushed to tolerance. Hot Celestin Vichy water was begun by mouth in small amounts after the stomach had had twelve hours of absolute rest and gradually, but rapidly, increased. On the fourth day orange juice was added. On the fifth day from operation her diet was resumed,—two orange albumens, half a grapefruit, 80 gm. fat,—and from that time it was very gradually increased. Through a slight inadvertence, on the sixth day after operation, she took 150 gm. of fat with 47 protein, and 20 carbohydrate, and showed only a very slight ferric chloride reaction, and a negligible Benedict. There was a strong Benedict reaction immediately after the operation which became practically negative in four days. On the eleventh day after the operation the stitches were removed, the stump being well healed. The patient was in good spirits, with sparkling eyes, and a smile on her face, satisfied with her diet and everything else. She was taking, protein 56, fat 104, carbohydrate 34, adding 10% of the fat and 58% of the protein, give a potential carbohydrate of 77. The total calories were 1330. Her blood pressure 164/80, her pulse 90 and regular. The Benedict and ferric chloride tests were absolutely negative. The patient was evidently drawing from her endogenous fats in addition to the 1300 exogenous calories, as it takes more than 1500 calories to produce a smiling satisfaction on the face of a patient.

During the days immediately following the operation, this patient took over 4400 cc. of fluid by the mouth daily, besides occasional enteroclysis. The urine averaged only about 1500 cc. daily,

but because of the absence of any evidence of renal irritation and because of the profuse sweating and the very good condition of the heart, as shown by a stationary blood pressure, regularly falling pulse and absence of edema, and because the patient was steadily gaining in strength and spirits, I deemed it wise to keep up the intake of fluid. I felt this was justified for the more rapid elemination of toxins from the body, especially as there was a moderately positive ferric chloride reaction for ten days after the operation and I did not wish to cut down the caloric intake. The temperature did not go above 99°, or the pulse above 100 after the operation. One-sixteenth grain of morphia was all that was given after the operation, although the patient had required codeine or morphia daily for some time previous to the operation.

Before the operation it had been impossible to satisfactorily get the patient's pulse in the left wrist. After the operation the pulse in the left wrist became clearly palpable and has continuel so.

Patient left hospital May the sixteenth, weighing about 150 pounds and showing a slight gain from day to day. Urine free from sugar and diacetic acid. Diet: protein 74, fat 162, carbohydrate 56. The fat is to be reduced and an effort made to keep the weight about stationary. On account of a tendency to neuritis, and because of the difficulty to get blood from this patient for blood sugar estimates the carbohydrate ration will be cut for the present.

1. Rettger, Leo F., Ph.D. and Cheplin, Harry A., Ph.D.: Bacillus Acidophilus and its Therapeutic Application, Arch. Int. Med. 29:357, March 15, 1922.

PELVIC PATHOLOGY AND THE CARDIO VASCULAR SYSTEM.

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This paper deals with women. Except for the acute infectious diseases a woman with a well pelvis is a woman well all over; a woman with a sick pelvis is a woman sick all over. Most of the morbidity of women relating to her nervous system, general health and well being revolves about her pelvic organs and their proper functionating. This statement must be accepted in a broad sense and includes not only the pelvic organs but her thyroid, mammary, pituitary and adrenal glands and indeed her entire endocrine system all of which are interrelated and interlocked and function together.

The blood pressure of the average young woman of properly functionating glands is as a rule lower than that of men. If there be excessive thyroid or ovarian action the blood pressure is still further lowered. If this action result in prolonged and profuse menstruation there may follow anemias and vitiated states of the blood, and a consequent poor supply of nutrition to the heart muscle itself through the coronary artery. The heart muscle degenerates and slight forms of dilatation with murmurs of a hemic type supervene.

As the average woman grows older and the ovary and other glands slow down in their action there is an accompanying advance in blood pressure. As the menopause is reached and the ovaries cease to functionate there is often an excessive rise in blood pressure with varying nervous symptoms which finally drives her to her physician. The discovery of excessive pressure leads the attendant sometimes to make the mistake of viewing this as cardio vascular disease of an organic nature. As a matter of fact it is not organic but purely functional and a sharp distinction must be made between this type of hypertension and organic cardio vascular change. Proper regulation of diet and habits, and the addition of thyroid

and ovarian feeding usually work wonders with the patient. These patients have been labelled "essential hypertension" by some in the profession for want of a better term to express physiological ignorance.

Pregnancy in most women is associated with a lowered blood pressure. This is due to the increased metabolism, the consequent increased drain on the thyroid gland and the associated diminution of the calcium content of the blood. If the thyroid fails to endure the strain of pregnancy and becomes exhausted metabolism is slowed down. The thyroid function is overcome by the hormones of the developing placenta and mammary glands. Elimination does not keep pace with metabolism. Toxins rapidly accumulate in the liver, the system is overwhelmed. Eclampsia follows with all of its serious and tragic effects. No woman with a good thyroid develops eclampsia. We must cease blaming the kidneys for this serious affection. The kidneys are only doing their best in the process of elimination. The albuminuria of eclampsia, the associated hypertension, headache and other manifestations are but the effects of a toxemia due to a thyroid breakdown, a failure in elimination often fulminating in its onset and tragic in its results.

The degenerations of the cardiac muscle associated with suppurating neoplasms of the pelvis are analogous to those observed in any other systemic bacterial invasion. The long standing pyogenic infections of the Fallopian tubes, pelvic abscesses, degenerating ovarian cysts or fibroid tumors as well as the cachectic toxemia of cancer in these organs must eventually affect the cardiac muscle. Hyaline degeneration of its fibres result from the toxemias of these conditions just as surely as they would from a pneumonia, an influenza or other infectious disease. Myocardinal weakness, dilitations, chronic nephritides and arterial diseases must eventually follow the one as truly as they do the other class of diseases if the pelvic pathology is allowed to continue indefinitely without relief.

The frequent association of fibromyomata of the uterus with cardio vascular changes, together with the fact repeatedly observed that the cardio vascular difficulty was usually modified or completely relieved following operative removal of the fibroid tumor as well as the startling fact that in most of the cases terminating

unfavorably the bad result was directly due to advanced cardio vascular changes rather than to death from purely surgical causes, has for a long time been a source of concern to surgeons and gynecologists.

Kasprozik in 1881 first called attention to the intimate connection of heart disease and fibromyomata. Rose in 1883 observed that in slow growing myomata secondary degeneration and atrophy of the heart muscle occurs. Hoffmeier studied at autopsy many of these hearts and found that they revealed fatty degeneration and brown atrophy of the myocardium. I will not weary you with all the references in the literature, but of many hundred of cases studied by several workers it was found that forty to forty-six per cent of the myoma cases analyzed, showed heart complications. The heart disease is characterized by relative insufficiency resembling a myocarditis but not entirely identical with it. In fibroids without abnormal bleeding brown atrophy was found while in fibroids associated with severe bleeding the condition was one of fatty degeneration of the myocardium.

It is of course going too far to say that all cases of cardio-vascular disease associated with fibroid tumors are due to the tumor, for there are other diseases and conditions that may upset the cardio-vascular system. Or indeed the cardio-vascular system may have been upset before the incidence of tumor formation. However, it must be admitted that the relationship between heart disease and myoma is of such frequency that the "myoma heart" must be recognized as a real entity.

According to Winter the cases most likely to be favorably influenced by the removal of the tumors are those dilatations and myocardinal degenerations due to prolonged hemorrhages and the consequent anemia. Considerable importance is attached by Cumston and Heywood Smith to the mechanical influence of back pressure on the kidneys and the resistance to the circulation produced in the hard unyielding substance of the tumor. It has been claimed by others that there may be a chemical basis for the changes in the cardio vascular system, a toxemia being produced analogous to that found in Basedow's disease, the degeneration of the tumor producing a protein toxin capable of affecting distant organs. Others have

mentioned the probability of a common cause of the two conditions producing on the one hand a fibrosis of the muscularis of the blood vessels and on the other fibromyomata of the uterus, thus suggesting that back of the whole question may lie a common endocrine thyro-ovarian disturbance. McClellan strongly favors the chemical basis as a toxemia arising from muscle extracts released from the tumor. This theory is supported by the experiments of Patta and Decio, 1911, who injected extracts of myomatous growths into lower animals and observed disturbances of the pulse, lowered blood pressure and cardiac irregularities.

At any rate the existence of the myoma heart and the associated renal changes as well as other conditions of pelvic pathology should be borne in mind by the internist, always, when treating women. The internist should not be content to go on treating impaired hearts and the cardio vascular system alone, if fibromyomata or other pelvic disease be present, but should aim to discover it earlier and provide for its prompt removal by surgery. The internist and the surgeon should work hand in hand and even after the removal of the trouble the patient should be kept for a certain length of time under the careful observation of the medical man until the equilibrium has been restored in the heart, kidneys and arterial system.

NOCTURNAL DYSPNEA.

Special Reference to Its Treatment with Thyroid.

By ROY D. ADAMS, M.D.

Dyspnea is the most important symptom of a variety of pathologic conditions. The majority of cases, however, are contributed by disturbances of the heart, lungs and kidneys. Often two or three of these organs are so extensively involved that immediate responsibility for dyspnea can be fixed only with the greatest difficulty, if at all. Recognizing the rôle played by disease of the lung and kidney in the production of dyspnea to be an important one, the present discussion is confined to those cases in which the cardiovascular system predominates the picture.

In heart disease, air hunger may present itself in three ways. It may be persistent even under conditions of prolonged rest. It may be incidental to moderate exertion. It may occur only in paroxysms. In the first instance it is associated with broken compensation. In the second, it expresses a limited cardiac reserve. In the third group, however, the every day laws of compensation and reserve apparently are not operative. Paroxysms are especially prone to occur during rest, not infrequently in the early morning at a time when demands upon the heart have, for some hours, been reduced to a minimum.

Paroxysmal manifestations are a common occurrence in severely decompensated cases. The term, as used here, does not refer to patients with evident continued dyspnea of the Cheyne-Stokes variety or exertion dyspnea of the paroxysmal type. It is confined to individuals capable of exertion without respiratory distress, with no gross secondary manifestation of heart disease and possessed of a vital capacity within normal limits.

The difference between the first and second groups of dyspneac patients is of degree rather than kind. The tendency is for the second, sooner or later, to merge into the first. The observations of Peabody and his co-workers on vital capacity and minute volume

have placed in our hands a means of estimating the reserve strength of the heart in these two groups, comparable to the measurement of thyroid activity basal metabolism. Whether or not venous engorgement of the pulmonary vessels and consequent stiffening or loss of elasticity of lung tissue is a satisfactory explanation for the mechanics of cardiac dyspnea may be questioned. The value and utility of vital capacity and minute volume, however, are definitely established. Unfortunately, vital capacity records are of little value in pure paroxysmal dyspnea for the reason that lung volume and elasticity are little impaired.

That the paroxysma of nocturnal dyspnea are cardio-vascular in origin is evidenced first, by the absence of lesions of the kidney and other organs; second, physical signs of organic damage to the heart; third, the supervention of broken compensation, and finally, by postmortem findings.

Fifteen cases have been under more or less close observation for at least one year by the writer. Classified clinically, four had myocarditis, six aortic disease, and five arterio-sclerosis. The majority of *all* cases of nocturnal dyspnea are associated with these three diseases. In this series, all patients had nocturnal dyspnea. Paroxysms during the day were very exceptional. None had kidney diseases of any consequence as shown by urinalysis and phenolphthalein test. All the patients were ambulatory and none had given up or changed his previous occupation.

The history of case No. 6 is typical, in the essentials. Male, age forty-three, letter carrier. Family history unimportant. Had syphilis at the age of twenty-two, for which he received treatment for two years. Married at the age of twenty-eight, father of three children, all healthy, wife has had no miscarriage or premature labors. For about six months prior to examination on July 5, 1916, he had been disturbed by attacks of shortness of breath upon falling asleep. On the preceding night he had suffered eleven attacks before finally obtaining any rest. The day preceding the attacks had been routine. Nocturnal disturbances had been experienced at irregular intervals, and sometimes followed days or weeks of complete freedom.

On physical examination the arteries were not palpably

thickened. The pulse was regular, synchronous in the radials and of the water-hammer type. There was not visible capillary pulse. The apex beat of the heart was in the nipple line under the sixth rib. There was no thrill. No dulness in the second interspaces. A soft diastolic murmur was audible on immediate ausculation at the aortic area and also from this point downward and toward the left.

The systolic pressure was 135, the diastolic 50. Otherwise the examination was negative.

The important clinical features were: first, history of syphilis; second, physical signs of aortic disease; and third, distressing nocturnal dyspnea which constituted the only symptom of ill health. Careful questioning in the fifteen cases and personal observation in some, revealed the sequence of events recorded in this case to be the typical picture. During a period of apnea or immediately preceding one, the patient would fall asleep.

The apneic period terminated in hypernea of the Cheyne-Stokes type. During the latter, consciousness returned with a most unpleasant and often alarming sense of air hunger. Examination during an attack showed no evidence of cardiac dilatation and little change in rate. If the threshold of sleep was successfully passed, three or four hours rest almost always ensued.

Reference, at this point, to the intensity of slumber curve supplies an interesting suggestion. Howell, Michelson and others have shown that the greatest intensity of slumber, as measured by the intensity of auditory stimulus necessary to arouse consciousness, is reached about an hour after the beginning. From this point there is a rapid fall in the curve and from the third hour on slumber is very light. As Howell expresses it, "The activities of the brain lie just below the threshold of consciousness."

The paroxysm occurs before deep slumber has been attained or after light sleep has supervened, only occasionally in deep slumber.

If we knew something of the physiology of sleep, a rational explanation might be offered. Unfortunately we do not. In a review of the information and theories on the subject by Haberman, the conclusion drawn is that "From the evidence so far accrued we may at least assume one can sleep whether 'product fatigued' or not and whether there be anemia or plethora in the brain and whether the

plethora be arterial of venous. One may sleep with lowered or hightened intra-cranial pressure, though the latter is (pathologically) especially sleep tending. Sleep does not depend upon hypo or hyper metabolism, though the former (in the sense of lessened energy denotation), no doubt more often obtains, and however endocrine dysfunction might induce or hinder sleep, regular sleep is nevertheless present in all types of endocrinopathy. There is no sleep center, nor is sleep occasioned through subcortical block."

Whatever the mechanism, it is apparent that the respiratory center is relatively unstable in "threshold slumber." This is shown by the great frequency of Cheyne-Stokes breathing during light sleep of normal individuals and by the tendency of paroxysmal dypnea to manifest itself at this time. It is as though during threshold slumber a conscious and a sleep control of respiration were tossing responsibility from one to the other.

The pathology of nocturnal dyspnea is the pathology of myocarditis, of aortic disease, of arterio-sclerosis and less frequently, of other forms of heart disorder. Recently, Leo Hess has reported autopsies on cases of cardiac dyspnea. I quote from an abstract of his article. "Three cases with the usual picture of continuous respiratory distress had in common a high grade of damage to the whole heart muscle, while on the coronary vessels no gross changes were demonstrable. In contrast were the anatomic findings in six patients with paroxysmal attacks of dyspnea. In all there were infarcts in the musculature of the left ventricle, chiefly at the apex, corresponding to the end branches of the coronary arteries, while the nonproximal portions of the muscle were completely free."

Hess discusses the possibility of connecting the anatomic findings with the clinical picture of shortness of breath. The dyspnea attacks he believes to be due to spasms of the pulmonary vessels, since in similar clinical pictures he often found an irregular distribution of blood in the various parts of the lungs, postmortem. This view, in regard to the participation of the pulmonary vessels in paroxysmal dyspnea, would have the advantage that it could explain the frequently concomitant pulmonary edema in cardiac asthma, for which the diminution of the power of the left ventricle cannot alone be made responsible."

Not questioning the observations of Hess, it is evident that his postmortem reports are of end results and not necessarily applicable to the relatively benign manifestation of disease here considered.

In following the subsequent history of the cases under observation for several years, the writer has become impressed by the frequent development of cerebral arterio-sclerosis. The types of cardiopathies exhibiting this symptom lend further evidence to this view. The conclusion that nocturnal dyspnea is an early manifestation of cerebral arterial disturbance in the bulbar region and incidentally in the respiratory center seems reasonable.

Treatment. In the treatment of noctural dyspnea dependent upon a diseased cardio-vascular system, general management of the patient and intelligent care directed toward the specific cause, are as important as in the usual cardiopathies. However, we are interested here in the relief of a symptom, precocious in its appearance and exhibiting peculiarities of its own. Therapeutic measures commonly employed are of two kinds. Curiously, these agents are physiologically antagonistic. One group is a cerebral depressant, the other an excitant. Peabody gets best results from morphia, Hoover from caffein citrate. In our experience, cerebral depressants aggravate the condition, unless pushed to such a degree that the patient is narcotized to his distress. While cerebral excitants, in the form of caffein, often prevent the paroxysm by keeping the patient awake.

In the present hey day of hormone therapy and endocrine romance, one hesitates to add a single indictment to this already crowded docket. In extenuation of the present offense the following may be offered: First, the administration of thyroid gland to patients with nocturnal dyspnea has afforded relief after other measures have failed. Second, the physiological action of thyroid upon metabolism, upon the excitability of the respiratory center and upon arterial vaso-motor tone in such as to indicate the rationale of its application to this condition. Third, the ultimate relief of symptoms following the administration of the iodides may be ascribed to stimulation of the thyroid through their iodin content. Finally, nocturnal dyspnea is most frequently encountered in patients at an age with which diminished thyroid secretion is concomitant.

The average age of the fifteen cases here reported is fifty-two.

The youngest is forty-two, the oldest, sixty-four. All patients were given dried thyroid gland. The average dose was about one and one-half grains three times a day. This dose was usually administered for from two to three weeks followed by an intermission of from one to two weeks. Twelve of the patients were distinctly relieved of dyspnea and showed no bad effects of the medication. Two were unaffected by treatment. One was unfavorably affected. He had increased dyspnea, rapid heart action and became nervous. The three patients who did not show improvement were of the myocardial group. The pulse rate of each was about eighty before institution of treatment.

It has been my experience that patients presenting paroxysmal dyspnea dependent upon myocarditis and having a material increase in pulse rate, react badly to thyroid. Administration of physiological doses to patients with marked cardiac damage is a hazardous procedure. Under any circumstances, patients receiving thyroid for the relief of nocturnal dyspnea should be under careful observation.

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DISCUSSION.

Dr. Rochester: I was very greatly interested in this paper, and I want to suggest one thing which may be a cause—I have had an opportunity, this year, to study a great many of the returned veterans, and a large number have suffered from shell shock, and a great many of them present this symptom of nocturnal dyspnea, and in most of those cases there is present gastric acidity, and giving of food when they wake up relieves that dyspnea, and that is what

I wanted to bring before you. I have not studied these cases over carefully as the reader of the paper, but I felt assured that we ought to investigate the digestive condition of the patient who suffers from it, because I find not only veterans but others that that symptom is of high gastric acidity, and by giving them food when they have this attack of dyspnea, they are relieved decidedly by it. I have no experience in the thyroid in these cases.

Dr. Babcock: I want to thank him for his suggestion in regard to the thyroid gland in connection with nocturnal dyspnea.

I had under observation a man who had more or less dyspnea during the day, but his paroxysms of dyspnea at night was most distressing, absolutely nothing I could do. The administration of morphine or any of the agents I had been accustomed to use—nothing of that sort gave them any relief for more than a very short time, and I was very happy to have this suggestion given me of the thyroid gland in these cases.

I use thyroid once in a while in certain cases, and with benefit when the patient has dyspnea on exertion.

I had one case under observation where it seemed beneficial, and for these most distressing paroxysmal attacks at night I would be very pleased to try it. I have seen a case which displayed dyspnea, and I have never seen anything do the patients any good, and I have regarded it as the gravest possible prognostic symptom.

Dr. Perkins: Since my paper of last year I have been carrying on the study further in regard to thyroid in relation to asthma, and my results are different from Dr. Adams, but it is probably due in part to the difference in ages of the patients. I have not separated distinctly the patients of about forty years of age, and some of those things, but I have seen quite a number of cases of asthma with paroxysm of attack at night in younger patients, where the attacks have been due to too much thyroid, the over-function of it. I have had cases where I have been able to demonstrate clinically that the paroxysmal attacks came, and it gave no indication whatever in regard to the cause of the attacks. But my experience also has been that it is dangerous, as I have seen patients come to me from other physicians in giving thyroid to some of the patients with dyspnea, because it is not so frequently due to hyperthryoidism, and I would caution you exceedingly against giving thyroid unless they are shown to be already hyper.

The President: Dr. Levine.

Dr. Levine: Nocturnal dyspnea of the kind just discussed apparently, in our experience in Boston, occurs in the same group of patients. In that general group of men there are some who sometimes at the age of fifty have cardiac sclerosis and arterial sclerosis, and the two types of drugs help, as explained by one of the speakers; one depresses the individual, so that they do not mind the discomfort. I do not think that those patients have a normal wind power during the daytime. That may not be their complaint to you. They may complain of nocturnal dyspnea, but I doubt on questioning them whether they have normal power, and if they would walk upstairs they would get winded; when

they are resting and have no discomfort they have—I do not recall any experience in some of the cases which have been discussed, but they are not hyperthyroid cases at all. From a practical point of view it can practically be overlooked. It would not be necessary, I do not believe, to make tests; they have a slow heart rate, most of them have a slow heart rate, and from a physician's point of view that is a handy test. During the day the heart rate while sitting on a chair would be 70, and it is apt to be regular. That for practical purposes rules out hyperthyroid.

CERTAIN TYPES OF AORTIC REGURGITATION.

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In the course of routine physical examinations of three hundred and fifty-five ex-service men, who had applied for compensation for ill health which they believed had been incurred in the military service during the World War, seven cases were seen which presented heart murmurs of the character usually connected with the murmur of aortic insufficiency or regurgitation. The following is a composite description of the murmur: It is diastolic in time and usually occupies the whole of diastole. Its maximum intensity is heard in the second and third left intercostal spaces. It is transmitted along the left border of the sternum, sometimes to the ensiform cartilage, sometimes to the fourth or fifth interspaces only. It may be heard in both the standing and the recumbent postures; and sometimes, but not always, it is louder in one position than in the other. It does not, as a rule, vary with the phases of the respiratory cycle. It is not much, if at all, influenced by exercise. It is constantly present.

The patients complain of dyspnea and palpitation with nervousness and sometimes of subjective arrhythmia. There are none of the attendant physical signs of aortic disease: no left ventricular hypertrophy, no Corrigan pulse, and no capillary pulse. The systolic blood pressure is normal or low and the fifth point is well above zero.

The questions to be decided in this group of cases are: (1) Does this murmur indicate regurgitation through the aortic valves or (2) Is it a functional murmur of the same class as the hemic systolic murmurs that are so common? (3) Does this murmur in the absence of cardiac hypertrophy and dilation warrant a diagnosis of aortic regurgitation with its attendant prognosis? (4) Should the patients who present this murmur be treated as cases of cardiac disease?

Our understanding of aortic regurgitation is that it is the one valvular defect that is attended by hypertrophy and dilation, by Corrigan pulse and capillary pulse, and by a blood pressure in which,

by the auscultatory method, the fifth point is often zero. Furthermore, the patients almost always give definite histories of antecedent rheumatic fever or some other so-called rheumatic disease, (chorea, tonsillitis, torticollis), or of syphilis.

TABLE I.

One of the patients had had rhumatic fever at the age of twelve or fifteen years, at least twelve years before he reported to us for examination; a second patient of the group had had several attacks of tonsillitis and one attack of rheumatic fever in 1918; a third had had an "occasional sore throat." The other four had had neither rheumatic fever nor tonsillitis; none admitted having had syphilis; none presented a positive Wassermann reaction. On physical examination none presented definite cardiac hypertrophy. In one case at two out of four examinations the oblique diameter of cardiac dulness was slightly increased over the figure that we have taken as high normal for that diameter (15 cm.) In another case a questionable increase was recorded at one out of three examinations. In no case did the electrocardiographic examination show left ventricular or right ventricular preponderance. In one case a capillary pulse was demonstrated at each of three examinations. Corrigan pulse was observed in one case at one of three examinations, and in three others it was questionable at one time or another, never positive. The fifth point of the ausculatory blood pressure estimation was at zero in one case once out of six examinations; in another case once out of three examinations.

The discovery of the murmur of aortic regurgitation without the attending physical signs of that disease led us at first to believe that these patients were examples of an adventitious diastolic murmur of functional character. Hirschfelder[7] says that the possibility of functional aortic insufficiency must be borne in mind by the clinician; but that it can rarely be verified and that the clinical diagnosis is hazardous. Fahr, Carter, Cosgrove and Edgar[4], in examining ten thousand troops found four recruits who presented diastolic murmurs which were very soft, usually heard only in the early part of diastole, and usually influenced by respiration, nearly disappearing in inspiration. White[11] says that a diastolic murmur

beginning early and best heard down the left border of the sternum, associated with the water-hammer pulse is pathognomonic of aortic regurgitation. This murmur may also be heard at the apex. Gittings and Smith[6], as a result of work done at U. S. Army General Hospital No. 9, say: "In the absence of the concomitant signs usually found in this disease, the interpretation of a definite diastolic murmur at the left sternal border is open to some question; but can hardly be decided on any basis than that of aortic insufficiency. Apparently this soft diastolic murmur is very frequently missed." Trémolieres and Caussade[10] are of the opinion that the murmur of aortic regurgitation, when heard at the left of the sternum, indicates that the case is recent one, and that in old cases only is it heard to the right of the sternum. Butler[3] says: "The presence of an aortic diastolic murmur is, in the majority of cases, reliable evidence of aortic insufficiency. But, even with this murmur, it is not safe to make the diagnosis without the confirmatory evidence of a water-hammer pulse, throbbing carotids, capillary pulsation, and a pistol shot sound in the femorals. If, however, the murmur is indistinct and is backed by a history of acute rheumatism in a young person, or of syphilis in a man of thirty-five or over, it is right to give the patient the benefit of the doubt and make the diagnosis on the murmur *per se*." King[8] says: "The murmur of aortic insufficiency is not simulated frequently enough by accidental or cardiorespiratory murmurs to warrant question of the diagnosis, provided the typical murmur is present." Bret[2] maintains that a basal diastolic murmur may be functional and quotes Devic and Bouchut in support of the contention. He admits, however, that as soon as one suggests that an aortic insufficiency is not the result of a lesion of the aortic orifice many objections are raised. He reports seven cases of functional aortic insufficiency and says that it is a condition that may be quite frequent in the cardiac disorders of patients with hypertension. Then he concludes: "Observed at the bedside, a Corrigan syndrome not emphasized (*souligne*) by a true diastolic hypotension ought rather to incline the diagnosis towards the hypothesis of a functional trouble." Gallavardin[5] has reported seven cases of aortic insufficiency in which the patients did not present a history of rheumatism and he says that the absence of such a history may mislead the

observer into an assumption of the existence of syphilis (unwarranted).

The determination of hypertrophy of the heart by percussion is well known to be open to many errors. Fahr, Carter, Cosgrove and Edgar[4] point out this fact and make the following statement: "The determination of hypertrophy of the left ventricle is fraught with uncertainty in many cases of percussion and position of the apex beat are the only means for determining the left border of the heart." Mackenzie[9] points out that there may be little or no dilation in certain cases of aortic regurgitation; and King[8], in a paper based on the examination of between 1,500 and 2,000 healthy soldiers makes the following statement: "One sees many soldiers with aortic insufficiency who present no demonstrable hypertrophy of the heart, nor indeed, any other secondary sign of the lesion. These men, also, frequently do not present symptoms, their condition being discovered accidently in the demobilization, after they have done full duty."

Cotton[12] as the result of a study of fifty cases of aortic disease in soldiers at the Sobraon Military Hospital, Colchester, says: "The most constant sign by which we can recognize early aortic disease is a diastolic murmur. * * * The heart may be slightly enlarged; frequently, however, there is no increase in the area of relative cardiac dulness. The pulse is not collapsing; there is no conspicuous pulsation of the carotids or the brachial arteries; and the blood pressure range is within normal limits. * * * One frequently observes in electrocardiographing patients with aortic disease that hypertrophy is equally borne by the two ventricles, so that the normal relationship of the two is maintained."

Electrocardiographic Studies.

Method. The electrocardiograms were all made under identical conditions: the patients lay flat on their backs in bed and Williams's German silver electrodes were used. The total resistances varied between 500 ohms and 1800 ohms, all being within the limit of 2000 ohms[14].

The following table shows the various measurements and observations made upon the tracings of these seven cases:

Table II.

The following summary shows the chief findings in this series of tracings:

Cardiac Rate. The heart rate is found to be normal (below 80 with the patients reclining) in four of the cases. The other three cases show a simple tachycardia (Case 1103, rates 100 plus on two occasions; Case 1132, rate 93; Case 1365, rate 91). Of these latter three cases, two show a normal rate on one occasion.

Sinus Arrhythmia. This is present in all seven cases, marked in two (1229 and 1262); moderate in two (1103 and 1365) and slight in three (1052, 1132, 1291).

P-Wave. This is above the maximum normal height[16] in one case (1103), of longer duration than normal in two cases (1103, 0.12 sec.; Case 1365, 0.12 to 0.14 sec.) and bifurcated or notched in one case (Case 1229).

R-Wave. This wave shows notching or thickening in all three leads in two cases (1052 and 1262), in two leads in one case (1365) and in Lead II only, in one case (1132). In one case (1103) it is aboxe maximum normal height in all three leads at three different examinations.

P-R Interval. A slight delay in conduction between auricles and ventricles is found in one case (1229). The P-R interval of Leads I and III measures 0.21 sec., that of Lead II 0.23 sec.

S-T Interval. This is of longer duration than normal[13-15] in three cases (Case 1229, 0.31 sec.; Case 1262, 0.30 sec.; Case 1291, 0.32 sec.)

Conclusion. None of the cases shows evidence of preponderance of either ventricle. There are found to be numerous minor departures from the normal, none of which is common to all of the cases or even to a majority of them. It does not seem probable that any of the usual features found in these cases have any bearing upon the clinical findings.

Determination of Heart Size.

Method. The method suggested and perfected by Bardeen[1] for determining heart volume, was used in this investigation. Tele-

rontgenograms were made of the patients, the silhouettes were traced from the negatives and the areas were measured with a planimeter. The volumes were calculated from Bardeen's tables. The heights had been recorded with shoes on and the weights had been taken with shoes, socks, union suit and trousers. Height without shoes and weight without clothes must be used in computing heart volume by Bardeen's method. In order to make calculations comparable it was necessary to correct the heights and weights as follows: 1.25 inches was substracted from the recorded height to compensate for shoes and four pounds was substracted from the recorded weight to compensate for the clothing. This latter is based upon the following weights: trousers with belt, 1 lb., 5.5 ozs.; army shoes, 1 lb., 14.5 ozs.; medium weight socks, 1.5 ozs.; light union suit, 5.5 ozs., totaling 3 lbs., 11 ozs. The more important of these corrections is the height correction and this should be practically constant for all of the individuals.

Table III.

Three of the five cases show practically a normal weight for height (Cases 1103, 1229 and 1262). The other two cases show marked deviation from the normal weights for their heights. Case 1132 is 19% underweight and Case 1365 is 37% overweight.

Table IV.

Heart Volume versus Body Weight.

The ratio of heart volume to body weight varies from 21% to 46% above normal in four of the five cases, Case 1365 is 20% below normal. The actual significance of these facts must be decided with reference to the actual versus the normal weights for the heights of these individuals. From this standpoint it will be noted that the two columns of Table IV show an inverse ratio, i.e. three cases (1103, 1132, 1229) which show an increased heart volume for body weight are all below normal weight for height and the one case which shows a diminished heart volume when referred to body weight (Case 1365) is markedly overweight (37%). Case 1262 has the most nearly normal weight for height of the entire group.

Heart Volume versus Height.

All of the cases show a definite increase in heart volume when referred to height, varying from 12% to 30%.

Heart Volume versus Body Weight and Height.

Case 1103 shows the greatest cardiac enlargement of all, the weight being practically normal while heart volume shows an increase of 41% and 30% respectively when referred to body weight and height. This is evidence of marked enlargement.

Case 1132 has a heart volume which is increased 46% and 19% respectively when referred to body weight and to height. This patient is 19% under weight but even when allowance is made for this the heart volume is markedly increased.

Case 1229 has a heart volume which is increased 21% and 14% when referred to body weight and height. This patient comes within the normal range in regard to weight so his heart volume is definitely increased.

Case 1262 shows a heart volume increase of 22% and 19% respectively when referred to body weight and height. This patient's weight is the most nearly normal of the whole five so that we can conclude that his heart volume is markedly increased.

Case 1365 is the only one of the series which has a diminished heart volume relative to body weight. The heart volume is diminished 20% but the patient is 37% overweight. This patient should weigh 60 kilos (132 pounds). If his weight were normal for his height and his heart volume 696 cubic centimeters the heart volume would then be 9% above normal. The heart volume is increased 12% when referred to height. These facts would seem to indicate a borderline heart but one which is still within the range of normal.

Composite Silhouettes.

A study of the composite silhouettes shows some interesting facts. Three of the five outlines fall rather close together (cases 1132, 1229, 1262). The other two extend far beyond the lines of these three. One case (1103) falls far to the right and the other (1365) falls far to the left. The one falling farthest to the right is

the largest heart of the five (887 cubic centimeters); the one falling farthest to the left is the smallest heart of the series (696 cubic centimeters) and is probably normal for the individual.

Table V.

Total Transverse Diameter.

The fact that practically no reliance can be placed upon the measurement of the total transverse diameter of the heart silhouette as an index of enlargement is again emphasized by these composite silhouettes. The greatest total transverse diameter of the series is 16 cm. (Case 1365) which is found in a heart showing a normal volume. This heart certainly is of unusual shape and its total transverse diameter is 3 cm. greater than is usual for a heart of its volume (696 c.c.) and yet the heart volume is normal for the individual. The smallest total transverse diameter, 12.6 cm. (Case 1229) is found in a heart which shows definite enlargement.

Teleroentgenograms versus Electrocardiograms.

The case showing the greatest increase in heart volume showed the most unusual electrocardiograms. Three different examinations were made after intervals of nine and six months respectively and the curves were practically constant in character. The P-wave was high and broad, lasting 0.12 sec. and suggesting auricular hypertrophy. The R-wave reached enormous heights 32.5 mm. to 37 mm. and the T-wave was of curious contour. The telerontgenograms of this case shows great extension to the right, of the right auricular curve.

The only silhouette suggesting a possible left ventricular preponderance is that of Case 1365. This heart is of normal volume for the individual. The electrocardiograms of this case show no evidence of a left ventricular preponderance.

Conclusions.

We feel that the following answers should be given to the questions suggested at the beginning of this paper:

(1) This murmur indicates regurgitation through the aortic orifice.

(2) It is not a functional murmur.

(3) The murmur in the absence of hypertrophy and dilation warrants the diagnosis of aortic regurgitation and the attendant prognosis of that valvular lesion.

(4) The patients who present this murmur should be treated as cases of cardiac disease before the occurrence of broken compensation.

(5) Analysis of the few cases in this series seems to support the general belief that the telerontgenogram offers the only method of determining actual enlargement of the heart. The electrocardiograms showed no evidence of left ventricular preponderance in any of the cases and the telerontgenographic findings are in no instance at variance with these findings.

Case Records.

Case 1052.

A man, aged 21 years, a draftsman by occupation, was drafted in 1918 and volunteered for immediate service in the Motor Transport Corps. He was sent to Fort Sheridan, Ill. He had no foreign service. While he was at Fort Sheridan he was well except for a cold. About two months after his discharge from the army he had a number of boils on various parts of his body and in February, 1919, he had influenza, abdominal type, and was in bed a week. He had been sick ever since that time, complaining of getting easily tired, pyrosis after eating, vertigo, gas in the stomach and twelve attacks of nausea and vomiting. The vomitus was brown and watery and sometimes contained undigested food. In May, 1920, he had nosebleed. He complained principally of being easily tired and he also had headaches every week or two; frontal and temporal, sharp, without radiation, which lasted four or five hours; and dyspnea and palpitation of the heart on exertion and excitement.

The family history was negative except that one brother had heart disease following scarlet fever.

The previous history showed that he had had diphtheria in

1903, influenza in 1919, and an occasional sore throat. Further than that he denied illness of every description. He had had no operation and had suffered no injuries.

His habits presented nothing seriously out of the ordinary. He denied the use of tobacco and of alcohol. There was a tendency to insomnia and constipation. His appetite was capricious and he had stopped all exercise except walking because he was so easily tired.

Physical Examination: The man was normal weight for his age and height, presented some acne on his face, tremor of the eyelids and hands and athetiod movements of the tongue; both tonsils were increased in size and looked infected, and there was a small goiter; Wassermann reaction negative.

Blood Examination: Erythrocytes, 5,710,000; leukocytes, 7,520; ratio, 1-759; hemoglobin, 88% (Sahli); color index, 0.78.

Differential Count:

Polymorphonuclear neutrophiles	56.0%—4141
Lymphocytes	40.4%—3038
Large mononuclears	1.8%
Eosinophiles	1.6%
Basophiles	0.2%
	100%

Urine Examination: Three examinations of the urine were made, in one of which there was a trace of albumin, less than 0.02% (Tsuchiya). Otherwise the urine examination was negative.

Electrocardiographic Examination: Made by Dr. Harry D. Clough. No. 91, September 25, 1920. Tracing made with the patient lying in bed. Resistance 1200 ohms. A skeletal muscle tremor is present in Leads I and III obscuring the electrocardiogram somewhat.

Lead I. Average ventricular rate 65 per minute. A marked sinus arrhythmia is present. The P-wave can be definitely identified in only a few places owing to the skeletal muscle tremor which is present. The conduction time is at the upper limit of normal. A slight notching is present on the up-stroke of the R-wave. The S-wave is marked in this lead.

Lead II. The average ventricular rate is 68 per minute. A marked sinus arrhythemia is present in this lead as in Lead I. The auricular wave is poorly defined throughout the entire lead and can be identified in only a few places. The conduction time appears to be at the upper normal limit (0.20 second). The deflection time is about 0.08 second and there is a suggestion of shading of the down-stroke of the R-wave. The T-wave is well defined and positive.

Lead III. The skeletal muscle tremor greatly obscures this tracing. The average ventricular rate is 68 per minute. The auricular wave cannot be distinguished. The deflection time appears rather long (0.08 second) and the R-wave shows a deep notching on the up-stroke near its apex. Occasionally the notching occurs at the very appex of the R-wave.

Note. It is not possible to decide absolutely in regard to the P-R interval; it appears to be at the upper normal limit at least and it is possible that there may be some beginning delay.

Opinions are somewhat divided in regard to the basis for a diagnosis in interventricular conduction defect; but it is certain that these electrocardiograms are not normal and are sufficiently suspicious to warrant further electrocardiographic study.

Diagnosis: Marked sinus arrhythmia. Intraventricular conduction defect.

Case 1103.

A man, aged 27 years, a candy maker by occupation, was drafted in 1918. He was sent to Camp Devens and assigned to the infantry. He was gassed in a defensive sector in August, 1918, and was returned to the United States as a sick casual in November, 1918. He was discharged on an S. C. D. for cardiac hypertrophy and valvular heart disease, mitral and aortic insufficiency. After he was gassed he was blind and had respiratory symptoms with the expectoration of blood. Before he left the base hospital in which he was treated he began to have attacks of vertigo, palpitation, precordial pain and dyspnea, which had continued until the time he was first seen. Sometimes he would have eight or ten attacks a day; at other times he would go two or three days with none. The attacks were usually brought on by exertion. The precordial pain was dull, vice-like, without radiation and was felt to the right of the sternum

as well as over the precordium. He had had a few attacks of fainting and unconsciousness, irregular paroxysms of coughing without expectoration; no spontaneous dyspnea; no abdominal pain; some nausea; no vomiting. He was very nervous and despondent, although he denied the latter condition.

The family history was negative; he was one of three children; all living; and his parents were not nervous.

The previous history showed that he had had a mild attack of scarlet fever in 1903; acute articular rheumatism in 1905 or 1908 affecting his knees, hips and wrists. He denied all other diseases. He had been married in April, 1919, and his wife had had one healthy child. He had had numerous furnucles on various parts of his body; particularly in the external auditory canals.

His habits presented no excesses in the use of alcohol or tobacco; but he was not bathing with sufficient frequency nor was he taking sufficient exercise.

The physical examination showed that he was twenty-one pounds underweight for his age and height; the occipitofrontalis control was irregular; he could wrinkle his forehead and frown; but there was a lack of relaxation between the movements. Dalrymple's and Mobius' signs were present; there was a slight exophthalmus, lachrymation while the eyes were being examined, tremor in the eyelids, and harsh sounds over the posterior aspect of the right lung.

Wassermann reaction, negative.

Blood Examination: Erythrocytes, 5,130,000; leukocytes, 9,920; ratio, 1-517; hemoglobin, 88% (Sahli); color index, 0.85.

Differential Count:

Polymorphonuclear neutrophiles	78.8%—7746
Lymphocytes	13.2%—1309
Large mononuclears	3.0%
Eosinophiles	4.0%
Basophiles	0.8%
Myelocytes	0.2%
	100.0%

Urine Examination: Four examinations of the urine were made

all of which were negative except for a low specific gravity in two (1.014 and 1.013) and phosphates in two.

Electrocardiographic Examination: Made by Dr. Harry D. Clough. No. 32 September 14, 1920. Tracing made with the patient lying in bed. Resistance 1800 ohms.

Lead I. The average ventricular rate is 107 per minute. The electrocardiographic complexes are normal in form. The conduction time is normal (0.16 second). The T-wave is well defined and positive.

Lead II. The electrocardiographic complexes are unusual in this lead. The auricular wave is very high (average 3 mm.); the R-wave is enormously increased, measuring 36 mm.; and the T-wave is of peculiar form consisting of a positive deflection followed by a very slight decline which becomes immediately continuous with the following auricular wave. The conduction time is normal (0.16 second). The average ventricular rate is 103 per minute.

Lead III. The average ventricular rate is 103 per minute. The P-wave is well defined; the conduction time is normal (0.16 second). The R-wave here as in lead II, is unusually high, measuring 24 mm. The T-wave is diphasic.

Note. The electrocardiographic complexes of Leads II and III are unusual in form but show normal time relations. There is no evidence of ventricular preponderance, right or left.

Diagnosis: Simple tachycardia.

No. 287, June 29, 1921. Tracing made with the patient lying in bed. Resistance 1400 ohms.

Lead I. Average ventricular rate 75 per minute. The P-wave is positive but rather variable in its form from a pointed to a very rounded wave. P-R interval normal (0.15 second). R-1 measures 16 mm. T-1 is positive and measures 2 mm. A slight sinus arrhythmia is present.

Lead II. Average ventricular rate 68 per minute. This complex is very unusual in appearance. The P-wave is pointed and widely spread at the base; it averages 3 mm. in height. The P-R interval in 0.14 second. The R-wave is unusual in type, measuring 32.5 mm. in height. The T-wave is positive; but is poorly defined and appears to measure less than 1 mm. in height.

Lead III. Average ventricular rate 65 per minute. The P-wave is well defined and positive; conduction time 0.14 second. R-3 measures 17.5 mm. and the T-wave is diphasic.

Note. The time relations are normal and there is no evidence of preponderance; but the R and T-waves in leads II and III are unusual.

Diagnosis: No definite diagnosis can be made.

Electrocardiographic Examination.

No. 414, December 16, 1921. Tracing made with the patient lying in bed. Resistance 1500 ohms.

Lead I. Average ventricular rate 103. The P-R interval is 0.12 second. The T-wave is poorly defined and in place is definitely negative. R-1 shows a respiratory variation in amplitude; from 14 to 15 mm. in height.

Lead II. Average ventricular rate is 100 per minute. The complexes are all uniform, but are very abnormal in appearance. P-2 measures 3.5 mm. and is broad at the base. P-R interval measures 0.16 second. R-2 is of enormous amplitude, measuring 37 mm. T-2 is definitely diphasic.

Lead III. Average ventricular rate is 103 per minute. The complexes in this lead resemble in a general way those of lead II. P-3 is 2.5 mm. in height. The P-R interval measures 0.16 second. The R-wave measures 24 mm. with a slight respiratory variation. T-3 is diphasic. There is no evidence of preponderance of either ventricle.

Diagnosis. Tachycardia, simple (average rate 102). Average respiratory rate 25 per minute.

Radiologic Examination: Made by Dr. James M. Flynn. November 10, 1921. A seven foot plate made. Diameter of arch of aorta, 4.2 cm. Median right, 6.2 cm.; median left, 9.0 cm.

Otolaryngologic Examination: Made by Dr. E. S. Ingersoll. October 12, 1920. Ears: Drums normal. External otitis causing discharge. Nose: Septum deviated to left, cutting breath capacity 50%. Throat: Tonsils fibrous but not very much enlarged. Detritus in crypts. Tonsils should be removed. Naso pharyngitis with heavy mucus discharge.

Case 1132.

A man, aged 28 years, a chauffeur by occupation was drafted in 1918 and was assigned to the Motor Transport Service and sent to Camp Custer, Mich. He had no foreign service. While in the army he was well except for a sprained wrist. At the time of his discharge he was told that he had heart disease; but aside from having a little dyspnea when cranking his car he had no cardiac symptoms. Eighteen months after his discharge from the army he was complaining of dyspnea on exertion and a sensation of weakness in his left side, with nearly constant palpitation. Once in a while he would have an aching precordial pain; without radiation. There has never been the sensation of impending death. There were no other symptoms except an occasional nonproductive cough.

The family history was negative.

The previous history was entirely negative; the only one of the diseases of childhood that he had had was measles. He had had no operations and had had no other injuries than the sprained wrist already mentioned.

His habits presented nothing abnormal.

Physical Examination: The man was twenty-seven pounds underweight for his age and height; the occipitofrontalis control was lost and when he attempted to move the muscle he was able to wrinkle his forehead and there was a marked tremos in the muscle fibers, there was a tremor in the eyelids, enlarged tonsils, the posterior cervical, axillary, epitrochlear and inguinal lymphnodes were palpable, there was cogwheel inspiration at the base of the left lung and at the left apex below the clavicle, and there was no well marked dermographia.

Wassermann reaction negative.

Blood Examination: Erythrocytes, 5,870,000; leukocytes, 13,600; ratio, 1-425; hemoglobin, 100% (Sahli); color index, 0.85.

Differential Count:

Polymorphonuclear neutrophiles	58.6%—7969
Lymphocytes	34.4%—4678
Large mononuclears	2.2%
Eosinophiles	4.4%
Basophiles	0.2%
Myelocytes	0.2%
	100.0%

Urine Examinations: Twenty-one examinations of the urine were made in this case; eighteen of twenty-four hour specimens, and three of single specimens. The total quantity varied from 750 c.c. to 1900 c.c. The specific gravity varied between 1.016 and 1.035. A trace of albumin was found in one specimen. The urine contained a substance that reduced Fehling's solution in sixteen specimens; in three the Fehling reaction was negative, and in two it was questionable. Nylander's reaction was tried in eleven specimens; positive in five and negative in six. The fermentation test was tried in ten specimens: positive in three, negative in two, questionable in five. The questionable reaction consisted of one very small gas bubble at the end of twelve hours.

Quantitative determinations of glucose were made in four specimens, using Fehling's solution with the following results: 0.19%, equivalent to 3.61 gm. glucose in 1900 c.c. of urine; 0.159%, equivalent to 2.2737 gm. glucose in 1430 c.c. of urine; 0.586%, and 0.570%. Diacetic acid was never found.

Blood Chemistry: Examination made by Dr. John R. Booth. Blood sugar on three occasions: 0.093%, 0.186%, and 0.080%.

Electrocardiographic Examination: Made by Dr. Harry D. Clough. No. 119. October 14, 1920. Tracing made with the patient lying in bed. Resistance 800 ohms.

Lead I. Average ventricular rate is 93 per minute. S-1 is present. The complexes are normal in form. Conduction time is 0.16 second.

Lead II. Average ventricular rate is 93 per minute. The P and T waves are well defined and positive. P-R interval is 0.18 second. S-2 is rather prominent.

Lead III. Average ventricular rate is 93 per minute. The P and T waves are well defined and positive. The S-wave is prominent and notched on the down-stroke. Conduction time between auricles and ventricles is 0.18 second.

Diagnosis: Simple tachycardia.

No. 299. July 19, 1921. Tracing made with the patient lying in bed. Resistance 1300 ohms.

Lead I. Average ventricular rate 67 per minute. There is a slight degree of sinus arrhythmia. The complexes are all uniform and of normal appearance. The P-R interval measures 0.2 second. R-1 measures 10 mm. S-1 averages 3.5 mm. The T-wave is 2 mm. high.

Lead II. Average ventricular rate 71 per minute with a very slight degree of sinus arrhythmia. The P-R interval is normal, 0.2 second. R-2 measures 12.5 mm.; T-2 measures 4 mm. and is positive. There is a suggestion of a bifurcation of the P-wave, but it is difficult to be sure of it.

Lead III. Average ventricular rate 69 per minute with a slight sinus arrhythmia. The P-R interval measures 0.2 second. The beginning of the ventricular complex shows a marked M-shaped deflection. The T-wave is positive and measures 2 mm.

Diagnosis: Normal electrocardiogram, showing a slight sinus arrhythmia.

Note. There is no evidence of preponderance of either ventricle.

Rontgenologic Examination: Made by Dr. James M. Flynn. November 21, 1921. Diameter of arch of aorta, 4.0 cm. M. R. diameter, 4.1 cm. M. L. diameter, 9.2 cm.

Laryngologic Examination: Made by Dr. E. S. Ingersoll. November 23, 1920. Ears: Moderate retraction of the drums. Scar anterior right. Left normal. Function normal. Nose: Marked spur, high right. Deviation of septum to left. Patient has no complaint of inadequate breathing, but breathing capacity is 50% of normal. Throat: Tonsils large, fibrous. Seem quite clean at present. No evidence of absorption in cervical glands. Do not believe tonsils are causing much trouble.

Case 1229.

A man, aged 25 years, a telephone cable splicer by occupation

was drafted in 1918. He was assigned to an infantry regiment which was in the St. Mihiel offensive. He was neither wounded nor gassed. In training camp in this country he was well; but in the training area in France he began to get nervous particularly after long marches. One day he dropped out of the ranks and had a crying attack that lasted for an hour, and at the same time he began to notice excessive sweating. He was not excused from duty, but continued his hikes and continued to carry his pack. During the St. Mihiel offensive he was without food for three days, except for hard tack that was soaked in water. Then he began to have diarrhea with twenty-five to fifty stools a day, containing blood. He was hospitalized and was told by a medical officer that his diarrhea was due to eating food saturated with gas. He returned to the United States as a sick casual, class B2. At the time of his discharge the diarrhea had been cured, but he felt nervous. He returned to civil life and took up his old job, but had to give it up in eight months because it was too hard for him. He complained of being weak and nervous and two physicians told him that he had heart disease. The nervousness continued and he began to complain of aching eyes, blurring of vision, black specks and flashes of light. About four months before he was first seen he began to complain of precordial pain, which was sharp, like a knife, and radiated toward the abdomen. The pain was paroxysmal and occurred sometimes two or three times a day; sometimes he would not have an attack for a week. The attacks of pain were accompanied by sweating and weakness. About a year before he was first seen he began to complain of dyspnea and palpitation on excitement; there had been no spontaneous or nocturnal attacks.

The family history showed that the patient was the son of a mother who had died of carcinoma of the breast at thirty-eight; he had one nervous brother and two nervous sisters.

The previous history showed that he had had pneumonia and diphtheria in infancy, scarlet fever and septicemia in 1907; pleuritis in 1916; influenza in 1916 and again in 1918, and dysentery in 1918. He had two or three attacks of coryza and bronchitis every winter. In 1915 he had had a nasal operation. As a boy he had been thin and delicate.

His habits were regular with the exception of anorexia and insomnia. He was of the opinion that he slept only about four hours every night and was troubled by dreams in which people were getting killed, but he did not dream about the war.

Physical Examination: The patient was fifteen pounds underweight; his occipitafrontalis control was lost, pupils contracted; the lateral movements of the eyeballs were irregular and suggested nystagmus; there were tremors in the eyelids and hands, the posterior cervical, right anterior cervical, supraclavicular and inguinal lymphnodes were palpable. There was a small goiter with a bruit over both lobes, dermographia was unusually prompt, deep and persistent.

Blood Examination made March 26, 1921: Erythrocytes, 6,040,000; leukocytes, 8,560; ratio, 1-705; hemoglobin, 101% (Sahli); color index, 0.83.

Differential Count:

Polymorphonuclear neutrophiles	51.8%—4434
Lymphocytes	41.0%—3509
Large mononuclears	3.4%
Eosinophiles	3.0%
Basophiles	0.8%
	100.0%

February 25, 1922: Erythrocytes, 5,220,000; leukocytes, 7,280; ratio, 1-717; hemoglobin, 99% (Sahli); color index, 0.94.

Differential Count:

Polymorphonuclear neutrophiles	49.8%—3625
Lymphocytes	37.0%—2693
Large mononuclears	9.8%— 713
Eosinophiles	3.2%
Basophiles	0.2%
	100.0%

Urine Examination: Four examinations of the urine were made; two each of 24-hour specimens and single specimens. The total quantity varied from 560 c.c. to 1610 c.c. The specific gravity

varied from 1.014 to 1.028. Three of the specimens contained less than 0.02% albumin (Tsychiya). No glucose was found. Phosphates were present in one. No casts were found. Calcium oxalate crystals were found in one specimen.

Blood Chemistry: Examinations made by Dr. Joseph R. Booth. Two examinations were made four months apart. The nonprotein nitrogen was 35.0 mg. % in one and 37.0 mg. % in the other. Uric acid nitrogen was 2.7 mg. % and 2.8 mg. % respectively. Blood sugar was 0.10 and 0.08% respectively.

Electrocardiographic Examination: Made by Dr. Harry D. Clough. No. 290. July 1, 1921. Tracing made with the patient lying in bed. Resistance 1000 ohms.

Lead I. The average ventricular rate varies from 65 to 47 per minute, showing a rather marked sinus arrhythmia. The P-wave is positive, and the P-R interval is about 0.21 second. The R-wave is of low amplitude, measuring 5 mm. The T-wave is well dened, positive, and measures 2.5 mm. There is an S-wave which measures 3 mm.

Lead II. The average ventricular rate shows about the same degree of sinus arrhythmia as is present in the previous lead. The P-wave is positive, measures 1.5 mm., and shows an irregularity on the down-stroke. The P-R interval measures 0.23 second, showing a delayed conduction between auricles and ventricles. The R-wave measures 15 mm. The T-wave measures 6.5 mm. and is positive.

Lead III. The average ventricular rate shows the same degree of sinus arrhythmia as is seen in the other two leads. The P-wave is slightly diphasic, although in places it is definitely positive and in other places definitely negative. An irregularity is noted on the initial stroke of the P-wave. The R-wave measures 11.5 mm. The T-wave is positive and averages 4 mm. in height. The conduction time is difficult to determine on account of the character of the P-wave, but it probably longer than normal.

Diagnosis: Marked sinus arrhythmia. Delayed conduction between the auricles and ventricles.

Case 1262.

A man, aged 25 years, a laborer by occupation, enlisted in 1917

for the medical department. He went first to Columbus Barracks, and was transferred to Walter Reed Hospital where he served until he was discharged on a certificate of disability in 1918. At Columbus Barracks he was well and at Walter Reed Hospital he was well until he was admitted to the wards for tonsillitis with general muscular pain. His statement of his condition is: "The tonsillitis ran into rheumatism and the rheumatism left the leaking valve." At the time of his discharge from the army he was not complaining of symptoms. After his discharge from the army he worked on a farm, for a florist, for an electric manufacturing company, back to farm work and when he was first seen he was working for a cabinet making firm. During that period he had an attack of tonsillitis and tonsillectomy was done. At the time he was first seen his only complaint was of gas in the stomach. The attacks occur three of four times a week and are not attended by nausea or vomiting. He has a little dull precordial pain at irregular intervals, without radiation and occasional dyspnea.

The family history showed one case of heart disease, three cases of nervousness, and one case of rheumatism.

The previous history showed that as a boy he had been sickly, complaining of stomach trouble; he had had several attacks of tonsillitis, acute articular rheumatism in 1918, tonsillectomy in 1920, and frequent attacks of coryza. He had had no injuries.

His habits presented nothing out of the ordinary. He denied the use of tobacco and alcohol. He took almost no exercise.

Physical Examination: The patient was nine pounds underweight; there was some acne on his back; the occipitofrontalis control was lost; Dalrymple's sign was present; there was beginning arcus senilis; the teeth showed five gold crowns, one of which was tender; the posterior cervical, axillary and inguinal lymphnodes were palpable, there was a small goiter with a bruit over the right lobe; in the left axilla there was an area of dulness at the fourth rib and in the fourth interspace, with cogwheel breathing at the base of the left lung; dermographia and exaggerated knee jerks.

Wassermann reaction negative.

Blood Examination: Erythrocytes, 5,870,000; leukocytes, 11,280; ratio, 1-520; hemoglobin, 102% (Sahli); color index, 0.86.

Differential Count:

Polymorphonuclear neutrophiles	60.2%—6790
Lymphocytes	27.8%—3135
Large mononuclears	2.4%
Eosinophiles	8.8%— 992
Basophiles	0.8%
	100.0%

Urine Examination: Two specimens of urine were examined: one passed at the time of the physical examination showed an alkaline reaction and a specific and a specific gravity of 1.012. The other a twenty-four hour specimen showed a total quantity of 1870 c.c., a specific gravity of 1.017, and uric acid crystals in the sediment.

Feces Examination: An examination of the feces showed neither ova, protozoa, metazoa nor embryos.

Electrocardiographic Examination: Made by Dr. Harry D. Clough. No. 269. Tracing made with the patient lying in bed. Resistance 1800 ohms. May 18, 1921.

Lead I. Average ventricular rate 67 per minute. A rather marked sinus arrhythmia is present, maximum rate being 73 per minute, minimum rate 63 per minute. The complexes are all normal in form. The P-R interval measures 0.17 second. The T-wave is positive and measures 3.5 mm. R-1 measures 7.5 mm.

Lead II. Average ventricular rate 67 per minute. The sinus arrhythimia noted in Lead I is very much more marked in this lead; maximum rate 68, minimum rate 48 per minute. The P-wave in this lead is rather poorly defined, but is positive in sign. The P-R interval is within the normal limit, 0.18 second. R-w measures 14.5 mm. T-2 is well defined, positive and measures 3.25 mm. The complexes are of normal form. The deflection time is 0.06 second.

Lead III. Average ventricular rate during the first portion of the tracing is 65 per minute, for eleven seconds. Following this there is a progressive increase in rate during two cycles and then a fairly constant rate of 91 per minute is seen during the rest of the tracing, about six seconds. Associated with the slow rate is an inversion of

the P-wave and a rather poorly defined diphasic T-wave. When the rapid rate is assumed the P-wave has become upright and the T-wave better defined, in places positive and at other points again diphasic. There appears to be a slight change in the conduction time associated with this change in rate: during the period of slow rhythm the P-R interval measures 0.16 second, whereas during the period rapid rhythm the conduction time is slightly prolonged to 0.18 second. R-3 is notched throughout, it varies in amplitude from 6.5 to 8.5 mm. These variations appear to be rhythmic and associated with respiration.

Diagnosis: Normal electrocardiogram showing a marked sinus arrhythmia.

Note: The changes in the complexes noted in Lead III are believed to be due to a migration of the pacemaker within the sinus node.

Roentgenologic Examination: Made by Dr. James M. Flynn. January 20, 1922. Diameter of the arch of the aorta, 4.3 cm.; M. R., 3.7 cm.; M. L., 9.4 cm. There is a slight increase in the left heart.

Case 1291.

A man, aged 27 years, a machinist by occupation, enlisted in 1918 for the Pioneer Infantry. He was sent to Camp Wadsworth, was in the Meuse-Argonne offensive. He claimed to have been gassed in November, combined respiratory and abdominal types, but did not report it because "someone told me that they would kill you in the hospital if you went there." He was in the Third Army and did duty in Germany, returning to the United States in June, 1919. He claims that he began to be dizzy as soon as he was gassed and that the dizziness had continued from that time till he was first seen, July, 1921. He said that the dizziness was worse in 1921 than it was in 1918; the attacks came on whenever he worked hard and were accompanied by palpitation and dyspnea. The attacks were not spontaneous or nocturnal. Since his discharge from the army he claimed to have lost a day or two each week on account of vertigo during the time that he was employed. The attacks of vertigo came on when he first got up in the morning, so that he had to lie down again. During the remainder of the day he would have one or two

other attacks, always when he was working hard and always accompanied by palpitation. He also complained of occasional dull frontal headache, radiating to the occipital region, tinnitus, bad taste in the mouth, occasional expectoration from the back of the throat, sometimes with blood, and gas in the stomach.

The family history was unsatisfactory; he had lost a sister with meningitis, and his parents, although living, were both sick.

The previous history showed that he had had from three to six attacks of bronchitis every year for several years and influenza, respiratory type, in 1918. He denied operations and injuries.

His habits did not reveal anything important: he smoked a few cigarettes; he had apparently used alcoholics freely while he was in France and Germany, but denied the use of any at the time of the examination. He seldom bathed and took no exercise.

The physical examination showed that he was sixteen pounds overweight, there were tremors in tongue and hands, but further than this no physical findings except those concerning the cardiovascular apparatus.

Wassermann reaction negative.

Blood Examination: Erythrocytes, 5,850,000; leukocytes, 8,080; ratio, 1-724; hemoglobin, 96% (Sahli); color index, 0.82.

Differential Count:

Polymrophonuclear neutrophiles	61.6%—4977
Lymphocytes	34.0%—2747
Large mononuclears	2.8%
Eosinophiles	1.2%
Basophiles	0.4%
	100.0%

Urine Examination: A specimen passed at the time of the original examination showed a specific gravity of 1.028 and contained phosphates and carbonates.

Electrocardiographic Examination: Made by Dr. Harry D. Clough. No. 298. Tracing made with the patient lying in bed. Resistance 500 ohms. July 16, 1921.

Lead I. Average ventricular rate varies from 60 to 68 per

minute, showing a slight sinus arrhythmia. The complexes are all uniform and normal. P-R interval is normal, measuring 0.17 second. R-1 measures 10 mm. T-1 is positive and measures 2 mm.

Lead II. The average ventricular rate is 60 per minute, although there is a slight sinus arrhythemia. The P-R interval is normal, 0.2 second. R-2 measures 14 mm. The T-wave is positive and measures 2.5 mm.

Lead III. The average ventricular rate is 60 per minute, with a slight sinus arrhythmia. The P-R interval is normal, 0.2 second. The R-wave varies from 2 to 5 mm. in height and shows marked notching in various parts of the deflection, sometimes on the up-stroke, sometimes at the apex, sometimes on the down-stroke. The T-wave is positive and measures 1 mm.

Diagnosis: Sinus arrhythmia.

Note: The R-T interval measures about 0.28 second in all three leads.

CASE 1365.

A man, aged 29 years, a railroad brakeman by occupation, was drafted in 1917 and assigned to the field artillery and later transferred to the engineers. While in training in the United States he was well. In the American Expeditionary Forces he was in the St. Mihiel and Meuse-Argonne offensives, but was neither wounded nor gassed. In September he fell off a box car and was told that he had sustained a rupture. He complained of precordial pain, dull and without radiation. He also complained of abdominal pain after being on his feet for an hour or so. He says that on one two two occasions he has tried to have his hernia operated on but has been refused on account of his heart which physicians have told him is diseased. He has irregular frontal headache, palpitation, and complained of his left thigh feeling dead when he stepped on the left foot.

The family history showed a sister having died of diabetes, and the father of pneumonia.

The previous history was unsatisfactory; he made claim that he had never consulted a physician in his life until he was in the army.

His habits showed an overuse of tea, six cups a day, and a corresponding diminution in water ingestion; he was a rather liberal user

of tobacco, smoking one and seven-eights ounces of pipe tobacco in two days, and bathing only once a week.

Physical Examination: The man was forty-five pounds overweight, presented Dalrymple's sign, slight exophthalmus, tremor in the eyelids, chronic pharyngitis, moderate hypertrichosis, and exaggerated superficial reflexes.

Wassermann reaction negative.

Urine Examination: An examination of the urine made at the time of the first physical examination showed a high specific gravity and phosphates. A twenty-four hour specimen showed a total quantity of 510 c.c., a specific gravity of 1.033, phosphates and amorphous urates.

Electrocardiographic Examination: Made by Dr. Harry D. Clough. No. 369. October 24, 1921. Tracing made with the patient lying in bed. Resistance 1200 ohms.

Lead I. The average ventricular rate is 91 per minute. There is a very slight sinus arrhythmia. The conduction time is normal, 0.16 second. R-1 averages 14.5 mm. T-1 is positive and measures 2.5 mm. The complexes are all uniform.

Lead II. The average ventricular rate is 88 per minute and there is a slight sinus arrhythmia. The P-R interval is normal, 0.18 second. R-2 measures 22 mm.; T-2 is diphasic.

Lead III. The average ventricular rate is 94 per minute with a slight degree of sinus arrhythmia. The Q-wave is unusually prominent in this lead, measures 4 mm. R-3 averages 8 mm. in height. T-3 is definitely negative and averages 3 mm. in depth. The P-R interval is normal, 0.16 second. There is a noticeable thickening on the downstroke of the R-wave in this lead.

Diagnosis: Simple tachycardia.

Surgical Examination: Made by Dr. Charles W. Hennington. The man has a right inguinal hernia.

Rontgenologic Examination: Made by Dr. James M. Flynn. Diameter of the arch of the aorta, 5.3 cm. M-R diameter 4.8 cm. M-L diameter 11 cm. Transverse diameter 15.8 cm.

Otolaryngological Examination: Made by Dr. Edward S. Ingersoll. Ears, normal. Nose, low spur left. Adequate breathing

space. Throat, chronically infected tonsils. Larynx, epiglottis thickened.

Diagnosis: Chronic tonsillitis, tonsillectomy advised.

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DISCUSSION.

The President: These papers are now before you for discussion.

Dr. Hall: I was exceedingly interested in the paper of Dr. Swan. I believe those cases did have a moderate aortic regurgitation. I have twice seen cases of ordinary aortic regurgitation follow the regular course. Both were proven by autopsy. We can see how those cases present the clinical features.

I think, however, one thing ought to be stated, and I ought to say that we do not see cases of leakages more frequently than in other cases, a relatively pulmonic regurgitation, because a considerable number of people have been sent to Colorado, not by members of the Climatological Association, with a diagnosis of tuberculosis. Many of those cases have a tremendous hypertrophy of the right side of the heart, and with the additional burden in the altitude where they have been, it begins to leak.

The President: Dr. Thomas, will you discuss this paper in regard to the cases of pulmonary insufficiency?

Dr. Thomas: I had occasion to watch several of these cases develop gradually, and it has also been my good fortune to see a number of cases I have not been able to follow from the beginning, but there is no doubt about these cases developing pulmonary insufficiency, as in the same class of cases of dilata-

tion of the arteries—the clinical signs do not remain permanent all the time; they will come and go. I have one case under observation now which presents marked signs of pulmonary insufficiency and at other times the pulmonary insufficiency will disappear. When they do appear secondarily, there is an indication of temporary beneficial effect to those patients. It seems to relieve the heart of pressure, and the clinical history of those patients is better.

THE PRESIDENT: Is there any further discussion on this paper? If not, Dr. Swan, have you anything to add?

DR. SWAN: I probably omitted any reference to the edeography of these papers on account of the shortness of time. These men were all young men, and had been in the army. I do not remember exactly, but I think the oldest man was 27 and the youngest 22 or 23. The murmur was typical of aorta revegetation; it was heard at the base, and was transmitted along the left border of the sternum, sometimes heard at apex and the cartilage, and sometimes both.

We did not consider the question of pulmonary insufficiency, because the murmur had such a classical transmission.

There was one case that came up after we had the paper about ready to present. There is no edeography in this case. One of the men had rheumatism about thirteen years before he went into the army. He was taken into the army and the murmur was overlooked, but when he came out he was told about it. He did not know anything about it until he came out of the army. One had attacks of tonsilitis, and this was one of the men who had rheumatism, but I felt I could disregard rheumatism and tonsilitis in these cases. They all denied syphilis, but they had negative Wassermann reactions, so until we got the x-ray evidence we felt it was questionable.

PREVALENCE OF GOITER IN GIRLS AS NOTED IN THE EXAMINATION OF 1500 ENTERING STUDENTS IN MILWAUKEE COLLEGE.

By J. GURNEY TAYLOR, M.D.

Living in the so-called Goiter Belt and observing a large number of goiters in the incoming students at the Milwaukee Downer College and Seminary during the past few years, has led me to group our findings in this paper as roughly gleaned over a period of eight years. Fourteen hundred twenty-five cases represent but half of those which should have been included in this survey, but the remaining half of our case records which were destroyed accidently would from one's impressions have been substantially the same.

Every student upon entering presented a complete history of previous physical condition with record of diseases contracted before entrance. This is brought by the student having been submitted either by the parents or the family physician.

A complete physical examination is given by us and an accurate record has been kept of same.

The thyroid has been closely and uniformly studied by us in the course of these examinations but our nomenclature as applied has not been as satisfactory as desired.

We have classified them as follows: firstly, those glands with a general enlargement of the entire gland; second, in regard to the lobe involvement; thirdly, with the isthmus alone involved.

Persistent thyro-glossal tracts have only been sought for during part of our examinations, and therefore are not included in our figures. The importance of uniformity in classification will add greatly in increasing the value of the data regarding the location of the involvement. The classification such as presented by Marine and Kimball in their reports of surveys of goiter in school girls is an excellent one, divided as it is into normals, slight, moderate and marked enlargements, adenomas and persistent thyro-glossal tracts.

Age	Number of Cases	Normal	Rt. Lobe	Lt. Lobe	Isth.	Gen.
13	10	7	0	0	2	1
14	32	24	0	0	3	5
15	57	35	1	0	8	13
16	92	65	1	0	15	11
17	242	182	5	1	33	30
18	473	314	11	7	90	53
19	297	202	8	3	48	36
20	144	85	7	2	27	25
21	57	38	2	0	10	7
22	21	16	0	0	4	1
Total	1425	968	35	13	240	182
Per cent	—	67.93	7.7	2.8	52	40

Age	Number of Cases	Tremor and Tachycard	Exoph.	Tonsils	Blood with Enlarg.	Pressure without Enlarg.
13	10	0	0	2	106-66	117-76
14	32	1	0	4	126-71	116-72
15	57	6	0	6	117-77	114-82
16	92	5	0	11	118-76	118-74
17	242	23	0	30	115-70	113-75
18	473	39	2	78	118-75	128-70
19	297	20	1	40	117-76	118-77
20	144	24	0	20	117-79	119-74
21	57	5	1	5	118-80	116-80
22	21	0	0	3	115-80	125-77
Total	1425	123	4	199		
Per cent	—	27	0.8	14		

Age	Total Normal	% Normal	Total Goiter	% Goiters
13	7	.49	3	.21
14	24	1.68	8	.56
15	35	2.45	22	1.54
16	65	4.56	27	1.98
17	182	12.70	60	4.2
18	314	22.0	159	11.09
19	202	14.28	95	6.64
20	85	5.95	59	4.16
21	38	2.63	19	1.34
22	16	1.19	5	.35
Total	968	67.93	457	32.07

State	Number Students	Number with Thyroid Enlargement	Percent
Wisconsin	783	290	37
Missouri	11	4	36
Michigan	72	25	34
Minnesota	58	19	32

Indiana	33	10	30
Illinois	195	62	30
Iowa	67	18	27
Arizona	8	2	25
Nebraska	12	3	25
Ohio	18	4	22
North Dakota	35	6	17
South Dakota	43	9	20
Tennessee	8	1	12
Mississippi	10	1	10
Montana	10	1	10
Pennsylvania	12	1	8
Colorado	14	1	7
Kentucky	3	0	0
Texas	4	0	0
Kansas	9	0	0
New Hampshire	4	0	0
Alabama	8	0	0
Massachusetts	3	0	0
New Mexico	4	0	0

Simple goiter develops most frequently during the formative periods, either during foetal life, puberty or during pregnancy. This fact has been clearly established. It has also been recognized that there is a marked increase in frequency among females, it being six times more frequent than in males.

The recent report of the War Department[1] has emphasized the higher incidence of goiter in certain parts of the United States. It has been extremely interesting to follow their figures relative to the examination of a group of men of selective age and a sex relatively less affected. A total of over 20,000 cases of goiter were regarded, simple goiter 11,971, exophthalmic goiter 8,647, giving a rate of three-fourths of one per cent of the population examined. It was furthermore shown that simple goiter was relatively more common than exophthalmic goiter but geographically was distributed over a smaller and well defined area. The extreme northwest of the Great Lakes basin presented the largest number of cases of simple goiter. Idaho, Oregon, Washington, Montana, Utah and Wyoming led with the greatest number, twenty-seven to fifteen cases per one thousand men examined. The second division about the Great Lakes, Wisconsin, Michigan, North Dakota, Minnesota, West Virginia, Illinois, Iowa, Indiana, and Ohio presented fifteen to five cases per one thousand men examined.

It is also interesting to view the figures presented regarding

exophthalmic goiter. Washington state has a ratio of 9.42 per one thousand men (nearly one per cent of the men examined), Wisconsin 7.94, Michigan 6.79, Oregon 6.37, Illinois 5.57, Ohio 5.08, and Pennsylvania 4.81. The least amount of exophthalmic goiter was found in the Gulf States.

Simple goiter was almost absent through the southern states from Virginia to Colorado. In the government report this clear cut distribution of goiter in the Northwest and Great Lakes basin is commented upon from the standpoint of the character of water supply, the mountain water, which is soft, in the former areas, and the hard waters of the Niagara limestone in the latter. However, the reports further draw the deduction that the presence of lime in the water or the mountain origin of drinking water are not to be considered as definite and sufficient cases of goiter.

In the recent analysis, goiter was found to be in higher percentage among the Scandinavians and Finns than among other races, but again a large percent of these men came from the Goiter Belt of Northern Michigan.

Levin[2] reported thirty per cent of thyroid enlargement in 583 soldiers examined who came from the Great Lakes basin.

Blankenship[3] reports on the goiter findings in 13,796 students entering the University of Wisconsin in a period of seven years. Thyroid enlargement was found in 3,784 cases or twenty-eight per cent and exaphthalmic goiter in .6 per cent or ninety-three cases. Of the number examined, there were only twenty per cent from the neighboring states, seventy per cent from Wisconsin and the remaining from other districts. He classified the age and sex incidence as follows:

Male: Simple unclassified enlargement 21.9%, exophthalmic .29%. Female: Simple unclassified enlargement 41%, exophthalmic 1.2%. Average combined age 19.9 years. Male (130) 19.1 years. Female (170) 20.4 years.

He has called attention to the interesting fact that the mortality statistics show for the five years given, an average death rate in Wisconsin from goiter as 2.4 per 100,000 against that for the entire United States over a like period of time as 1.6 per cent per 100,000.

He furthermore emphasized in a study of three hundred cases

of simple goiter selected from the student examinations that 95.6 per cent of these cases presented some abnormality of the upper respiratory tract or the teeth, which might affect the metabolic function of the gland.

We are indebted to Marine and Kimball[4] for extensive surveys among the public school girls of Akron and Cleveland. I have selected a table giving the percentage of the thyroid involvement simply as a matter of comparison of those in the age group which I have submitted.

Age	10-12		12-14		14-16		16-18		18-20	
	No. of Cases	%	No. of Cases	%	No. of Cases	%	No. of Cases	%	No. of Cases	%
Normal	530	65.08	521	41.32	460	40.35	156	34.44	21	28.77
Slightly enlarged	394	41.69	680	53.92	578	50.70	235	51.88	44	60.27
Moderately enlarged	21	2.22	59	4.68	98	8.6	60	13.24	8	10.96
Markedly enlarged			1	0.08	4	0.35	2	0.44		

D. C. Hall[5] reports 2,086 men at an average age of twenty years, five months, and 1,253 women with the average age of nineteen years, three months. In the men, 374 or 17.93 per cent of enlarged thyroid were present. Of these 272 or 13.03 per cent were clossified as perceptible, 92 or 4.43 per cent medium and 10 or .48 per cent were classified as large.

In the women, 294 were classed as perceptible or 23.45 per cent, 85 as medium or 6.79 per cent, nine as large or .7 per cent, a total of 30.94 per cent. His age group of women was above that submitted in my figures but it at least demonstrates the prevalance of goiter in the northwest district.

Notwithstanding the great amount of study that has been devoted to the problem, it may be stated with authority, I believe, that the ultimate cause of goiter is unknown.

The immediate cause, however, is the lack of iodine, which fact has been closely established through experimentation with animals and with humans and the results of the iodine administration to the school children by Marine and Kimball has undoubtedly established this treatment as one that is essential. In the belts in which goiter is epidemic, the enlargement of the thyroid gland may be regarded

as a symptom indicative of an increase in the iodine needs, or interference with the normal utilization of available iodine or experimental deprivation of iodine. The results is the same in any event—Goiter.

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THE PRESENT BASIC PRINCIPLES OF VACCINE THERAPY.

By J. H. TYNDALE, M.D.

LINCOLN, NEB.

The first cornerstone of vaccine therapy is this: Bacterial invasion of the organism is, at first, local. When microbes leave the reservation, even to the point of setting up a bacteriemia, they still select tissues in which alone they feel at home and may proliferate at pleasure and at leisure.

Two deductions result from these facts:

1. That bacteria having gained entrance to the system, have limited opportunities for growth.

2. That other fluid or solid tissues not selected by them unite for their destruction.

Cornerstone No. 2. Now such contemptuous ignoring of bacterial invasion may be rudely shaken in three different ways:

Excessive quantity of bacteria; a lesser number of exalted virulence; and, the presence of more than one specie of bacteria (mixed infection).

Cornerstone No. 3—forming the basis (the *raison d'être*) of artificial immunity, of vaccine therapy. Once bacteria are established in a given tissue—in the lungs, for example—and are proliferating, it is evidence that this tissue is not immune. Also, that subsequent arrest of their activity is not to be credited to the defensive powers of the local tissue, but to the aid furnished by the organism outside of that locality. This is self-defense, auto-inoculation, pure and simple, and is accomplished by the so-called antibodies. These substances are found in the blood serum and act in various ways; the bacteriolysins destroy the bacteria, the antitoxins neutralize the bacterial poisons, the agglutinins bring about a clumping of the organisms, and, last but not least, the opsonins act on the bacteria in such a way that they can be taken by the polynuclear leucocytes, a process known as phagocytosis. In other words, a leucocytic

invasion of the infected locality, so-called "artificial auto-inoculation" is, as the name implies, an artificial aid in which assistance still comes from within the body and is exemplified by Bier's appliances for inducing artificial hyperaemia, by means of which local proliferation of bacteria is arrested by inflow of exudate into the parts.

It is the contention of all practitioners concerned in the actual treatment of pulmonary consumption other than through laboratory verdict, that self-inoculation, however apparently successful, is ever a standing invitation to a recurrence. Hence, the ever-growing necessity for and use of artificial immunity—introduction of defenses from without the body.

Cornerstone No. 4 comes as a summary of preceding statements: For permanent arrest of bacterial invasion we made use of—(1) self-(auto) inoculation; (2), artificial auto-inoculation; (3) introduction of the bacteria in question, either living or dead—preferably the former.

Result aimed at in either case: To increase antibacterial reaction on the part of non-affected tissues to the extent of reconstruction with careful avoidance of destruction. The system is to be made responsive either to natural or artificial stimulus—not active reaction, but effective resistance only.

It becomes at once apparent that the real difficulties are encountered at this point. Nothing in the domain of medicine so well illustrates the maxim that we must treat the individual instead of the name of the disease. This provokes the natural question of what therapeutic rules exist at the present day.

1. Wright's Opsonic index, serviceable as it has been in determining intervals between vaccinations and the increase of successive doses, is not applicable in practice. The clinical index remains our guide.

2. Place no misguided reliance on either form of self-inoculation, natural or artificial.

3. Minute amount of toxins are capable, under perfectly normal conditions, of calling out a profound systemic response. A small pimple, a one-sided nasal catarrh are oftimes accompanied by high temperature and general malaise. On this fact is based the

therapeutic law that preponderance of general symptoms over the local manifestations, calls for attention to the former ahead of the latter.

4. The best results, therefore, are obtained by doses of vaccine so small as not to induce systemic reaction during the next few hours. Minute, not massive doses are to be employed.

5. Toxin tolerance of each patient is strictly and absolutely individual. The same holds good of intervals between doses.

6. Bacteria fenced in by capsulation are not harmless. Encapsulation produced by self-inoculation, as exemplified by open air treatment, is never dense enough to prevent the entrance of bacterial agencies.

7. Can we vaccinate with impunity where the diffusion of bacterial products has already caused active reaction on the part of the organization? Are we still stimulating the distant tissues to produce additional antibodies, or are we exhausting them? Prof. Adami says:

"Here I am inclined to the belief that the fatal event in the majority of severe infections is not brought about by exhaustion of the tissues in general, but by action of the circulating toxins on the medullary nerve centers, and that the sign of exhaustion of the tissues or paralysis of the same and inadequate reaction on their part is a lowering and not a rise of the body temperature."

And again—"This may, I think, safely be laid down: That on the one hand a sinking temperature accomplished by increased weakness of pulse and respiration and advancing 'toxic' state is a clear signal that the reactive powers of the patient are being overcome and exhausted, so that vaccination can only be harmful, and on the other hand if the fever be somewhat high, provided that the pulse is steady with no signs of cardiac weakness, then vaccination may safely be undertaken. The good results otbainable in these cases can no longer be questioned, and what is intersting is that the system evidently benefits from the slight temporary added rise of temperature which shows itself during the six hours or so immediatly following the vaccination. But hyperpyrexia is a contraindiction."

To be still more definite: The limit of permissible (construc-

tive) rise of temperature is 99.5°. Hyperpyrexia (destructive) from 99.8° to 100° and over.

In the near future I may be able to give my results in artificially keeping down hyperpyrexia—guaiacol externally, pyramidon internally—while keeping up the vaccination. Thus far the results do not promise well.

Lastly as to the quality of vaccines and their source: It goes without saying that an auto-bacterine is the ideal vaccine. Not unlike the opsonic index, it is beautiful and correct in theory, but hardly applicable in active practice. Serous auto-therapy is successfully practiced in cases of tubercular pleurisy with effusion, both by partial withdrawal of the fluid and its hypodermic reinjection into the connective tissue and by promoting its reabsorption.

Here, it must be remembered that this is passive immunization, on a par with the serums of Maragliano, and others, declared as having no specific value by the International Congress on Tuberculosis. At present, therefore, our reliance must be placed on stock vaccines, both human and bovine.

A NOTE ON THE PRACTICE OF ARTIFICIAL PNEUMOTHORAX BY THE HIPPOCRATIC SCHOOL.

By ALLEN H. KRAUSE, M.D.,

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It is quite likely that few of us ever open Thomas Young's book on Consumptive Diseases. Published in 1815, its complete title is *A Practical and Historical Treatise on Consumptive Diseases, Deduced from Original Observations and Collected from Authors of All Ages.* That the author's name is associated with tuberculosis should be a source of pride to all who take an active interest in the subject. The same man gave his name to "Young's Modulus" and the Young-Helmholtz theory of color perception. He discovered the mechanism of the crystalline lens and formulated the best theory of tides. The universality of his genius is attested by authoritative investigations on "shipbuilding, gas-lighting, standardization of the seconds pendulum and the imperial gallon, longitude and life insurance." He was a famous Egyptologist and the decipherer of the Rosetta Stone. Garrison characterizes him as "the most highly educated physician of his time," "probably the handsomest of all the great physicians,' 'and "one of the greatest men of science of all time." To Helmholtz he was "one of the most clear-sighted men who ever lived."

When Young wrote his book he was a Fellow of the Royal College of Physicians and physician to St. George' Hospital. In his Preface he states that he has tried to include "every fact of importance" which he was "able to observe, or find recorded, with respect to the nature and cure of the diseases belonging to a single genus." The book will not bore you. It will give you a new sense of how many of our modern methods and principles are only rediscoveries of what were put forward generations ago.

At page 117 one is startled to find Young mentioning a method of treatment for adhesive pleuritis which he refers to the Hippocratic School and which in every essential is that of artificial pneumo-

thorax. The citation sent me scurrying to the original source and in section 59 of the second book of "On Affections," as found in Littré's bilingual text of Hippocrates I found the passage mentioned by Young. We are so accustomed to date the inception of the idea of artificial pneumothorax from Forlanini's first work of forty years ago, that I deem this habit sufficient apology to rescue from an oblivion of more than two thousand years a distinct familiarity with the principle of the method.

In the section of Hippocrates referred to we read as follows:

92 DES MALADIES.

59. [1]Ὁ πλεύμων προσπεσὼν ἐς τὸ πλευρόν· [2]ἢν ὁ πλεύμων πρὸς τὸ πλευρὸν προσπέσῃ, βῆξ ἴσχει καὶ ὀρθοπνοίη, καὶ σίαλον βήσσεται λευκὸν, καὶ ὀδύνη τὸ στῆθος καὶ τὸ μετάφρενον ἴσχει, καὶ ὠθέει προσκείμενος, καὶ δοκέει τι ἐγκέεσθαι βαρὺ ἐν [3]τοῖσι στήθεσι, καὶ κεντέουσιν ὀδύναι ὀξεῖαι, καὶ τρίζει οἷον μάσθλης, καὶ τὴν πνοιὴν ἐπέχει· καὶ ἐπὶ μὲν τὸ πονέον ἀνέχεται κατακείμενος, ἐπὶ δὲ τὸ ὑγιὲς οὔ, ἀλλὰ δοκέει τι αὐτῷ οἷον ἐκκρέμασθαι βαρὺ [4]ἐκ τοῦ πλευροῦ, καὶ διαπνέειν δοκέει διὰ τοῦ στήθεος. Τοῦτον λούειν θερμῷ πολλῷ δὶς τῆς ἡμέρης, καὶ μελίκρητον [5]πιπίσκειν, καὶ ἐκ τοῦ λουτροῦ, οἶνον λευκὸν κεραννὺς καὶ μέλι ὀλίγον, καὶ δαύκου καρπὸν τρίψας καὶ τῆς κενταυρίης, διεὶς τούτοισι, διδόναι χλιαρὸν καταβρόφανειν· καὶ προστιθέναι πρὸς τὸ πλευρὸν ἐς [6]ἀσκεῖον ἢ ἐς βοείην κύστιν ὕδωρ χλιαρὸν ἐγχέων, καὶ ταινίῃ συνδεῖν τὰ στήθεα, καὶ κεῖσθαι ἐπὶ τὸ ὑγιὲς, καὶ τὸν χυλὸν διδόναι τῆς πτισάνης χλιαρὸν, καὶ ἐπιπίνειν οἶνον ὑδαρέα. [7]Ἢν [7]δὲ ἐκ τρώματος τοῦτο γένηται ἢ τμηθέντι ἐμπύῳ, γίνεται γὰρ, [8]τούτῳ κύστιν πρὸς σύριγγα προσδήσας, ἐμπιπλάναι τῆς φύσης καὶ εἰσιέναι ἔσω, [9]καὶ μοτὸν στερεὸν κασσιτέρινον ἐντιθέναι, καὶ ἀπωθέειν πρόσω. Οὕτω διαιτῶν τυγχάνοις ἂν μάλιστα.

FIG. 1.

The Lung Inclining Against the Side: When the lung inclines against the side, the patient has a cough and is orthopnoeic; the expectoration is colorless; and he has pain in the breast and back. The lung is thrust outward, pressed closely to the side. The sick man feels as though a weight were in his chest. Sharp stabbing pains assail him; a leathery bruit is heard; and breathing is restrained. The patient can endure lying upon his painful side but not upon his sound side, for it then seems to him as though a weight were suspended from his side and that he breathes through his chest . . . One should constrict the chest with a band, when the patient will lie upon his sound side . . . If this affection

results from a wound or, as sometimes happens, from an incision for empyema one should attach a pipe to a bladder, fill the bladder with air and send the air into the interior of the chest. And one should insert a solid pewter sound and push it forward. It is by this method that you will get the very best results.

Littré believes that by the expression "the lung inclining (or falling) against the side," Hippocrates means empyema,—that this was his name for the disease. He quotes Fournet to the following effect:

In some cases of operation for empyema and in a great number of cases of pleurisy, a pleuritic friction rub testified to a drawing together of the two pleural surfaces. In one of our patients it was a more certain sign than auscultation or percussion afforded: the lung was in contact with the sides; but its periphery was so markedly indurated that there resulted great obscurity of the chest tones and a marked diminution of breath sounds; and these two circumstances led us to the erroneous belief that the lung was kept at a distance from the side by a layer of fluid.

Littré continues that "without a doubt, in cases of operations for empyema and of opening the chest, it was the movement of approximation of the lung to the sides which suggested to Hippocrates the idea of calling this disease 'The Lung Falling Against the Side.'"

At any rate, the therapeutic method described is plainly that of inflating the chest with air, and we must believe that it was actively practised. Young's comment leaves no doubt that more than a hundred years ago he, for one, partially grasped the underlying principle of the procedure; for he remarks that "however extraordinary the practice appears, it is not impossible that it might be beneficial where the adhesion was not such as to prevent the collapse of the lungs, allowing the wounded parts to reunite more readily than in the natural process of respiration, since they would be more intimately in contact with each other, though not more at rest." Alas, the last phrase indicates that he missed the real rationale of the procedure. Perhaps, though, the ancients did not, but were alive to the fact that relative rest of respiration would result and to its importance. That they may have been is borne out by the advice in the same section to constrict the chest with external bands, though the context would seem to show that this was done

primarily to relieve pain. Yet this therapeutic aid is also one that is latterly being revamped in a variety of guises.

I am not at all clear as to what is meant when it is advised to insert a sound and push it forward, unless it be to push the lung away from the chest wall so as to allow the inflow of air.

In his schema of the Hippocratic Collection Francis Adams places all four books of the treatise "On Affections" among the "works certainly not written by Hippocrates," but "about contemporary with Hippocrates" and of unknown authorship. He remarks that "almost all the modern authorities . . . concur in rejecting the whole four as spurious." Dioscorides, the Commentator, believed that the second book, which contains the passage we have resurrected, was written by Hippocrates the Younger, the grandson of the Father of Medicine, and this opinion was shared by Galen. Littré placed the part "On Affections" among the writing of the Coan School, of contemporaries or disciples of Hippocrates. It seems certain, therefore, that the paragraph which we have had under discussion records a method in practice as early as the fourth century B. C.

LEGEND.

Fig. 1. Text of Section 59 of Book II of "On Affections." From Œuvres complètes d'Hippocrate, E. Littré, vol. vii, Paris, J. B. Baillière, 1851.

DISCUSSION.

The President: Is there any discussion on this paper?

Dr. Forster: As we all know, there have been quite a number of suggestions, I think, that 1818 was the earliest in literature, and as to that Dr. Krause called attention, but certainly an examination of the literature shows that quite a number of men whom conceived the idea of pneumothorax and its possible benefits—although possibly it was not carried to fruition until it was introduced, and we know how long it was after its introduction that it was generally accepted.

Dr. Krause calls attention to the use of the bladder of air in introducing air into the chest which makes me think of what an old doctor told me about his practice in following Murphy. Murphy used, I believe, a cylinder of gas or gas bags. This doctor weighed some 246 pounds, and he said he was quite young at the time that Murphy introduced his treatment, and that he found difficulty sometimes in getting the gas to flow in properly and he was accustomed to sit on the bag.

A BRIEF CLINICAL COMMUNICATION CONCERNING THE VALUE OF EARLY DIAGNOSIS AND TREATMENT IN THE SYNDROME KNOWN AS CHRONIC OR LATENT UREMIA.

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Introduction.

In bringing before this Association this brief clinical communication on the subject of the very interesting condition commonly referred to as uremia, I wish at the start to point out that the title given my paper in the program is somewhat misleading. I have not the slightest intention, even if time did permit, to deal with this interesting syndrome in any very comprehensive or thorough manner. My only purpose is to provoke a discussion and thereby encourage on the part of the clinicians and clinical investigators a more intensive and practical study of the as yet unkonwn etiology of the uremic syndrome and to suggest the advisability of training ourselves in future to study its varied course not only in its earliest course but especially in connection with the various other syndromes with which it is so often clinically associated. My conviction is that by doing this, that our search for its ultimate cause will be much sooner rewarded. I hope, as I proceed in my argument, to make the clinical point clear that it is often intimately associated with many other diseases and disease syndromes due to abnormal metabolic changes.

I am going now to digress a moment from my main theme to refer very briefly to a clinical suggestion made frequently by Sir James Mackenzie, i.e., that our text book writers are so prone to describe in minute detail the terminal stages of diseases, but give but scant attention to any attempt to describe diseases or disease syndromes at their earliest beginnings. He also pointed out as a reason for this state of affairs that such writers, mostly hospital physicans, could only describe and write about what they actually

saw and observed, hence, as they usually only saw the terminal changes of diseases, it was manifestly impossible for them to describe or to write about early symptoms. It has been frequently commented upon how much more real progress was made in the study of certain diseases, during the World War, by students of internal medicine than was made by surgeons. One illustration will serve to bring out this point. In one army camp during the war Dr. W. W. Herrick of New York had at one time, under his personal observation for study, more cases of epidemic cerebrospinal meningitis or meningococcus meningitis than any previous observer. By carefully observing all these cases he was able to establish the fact that some of his cases did establish an immunity to this disease in twenty-four hours, others in thirty-six or forty-eight hours, while not until the disease was quite advanced did his cases get meningitis. In the throats of all of his cases, however, he was able to demonstrate the presence of the meningococcus. He thereby established the very interesting fact that the case who had established a personal immunity to this disease in twenty-four hours was a carrier and the same was true of other early cases.

I would like to draw the parallel here that uremia should be studied from a similar early standpoint. It should, however, be emphasized that whereas Dr. Herrick knew the cause of the disease he was studying, we do not.

Two years ago the author of this communication, in a paper entitled "Blood vs. Tissue Acidosis," which he read before the American Therapeutic Society, called attention to the great clinical value in the early diagnosis of acidosis of Dr. Henderson's "Breath holding test." He would suggest that possibly an equally simple test for the diagnosis of early uremia might be possible. Hypertension is often found closely associated with uremia and a comparison between their respective courses may be suggested. Hypertension is also a condition in which the exact etiology is as yet unknown. It is quite generally admitted by good clinicians that it is due to the formation in the body of some toxic substance either bacterial or chemical and whose exact nature is as yet unknown. Its action on the body is at first slight and intermitten. Its effects comes and goes. It is at first easily controlled by rest and an appropriate diet, but as time

goes on and the effects of this unknown toxin still is exerted, the clinical picture changes, the acute intermittent stage disappears and a chronic more or less permanent condition sets in. A process which at first was merely functional and temporary now has become organic and permanent. The hypertension which was at first purely functional and temporary and unassociated with any marked anatomical changes is now transformed to a hypertension associated with more or less marked and permanent changes in the vascular system, resulting from the prolonged and the more or less destructive action of this unknown toxin. There is now no doubt that, at this stage of the trouble, that this hypertension is a more or less necessary compensatory mechanism on the part of nature to force the blood through rigid, narrow and diseased vessels, a condition which is well illustrated in the latter stages of chronic interstitial nephritis and in the general arteriosclerosis, the result of age.

Dr. Elsner, in summing up his ideas concerning the etiology of uremia says, "Whatever theory we finally adopt to explain the production of the uremic syndrome, must always include evidence of faulty metabolism and in changes which reduce the functional capacity of the kidney." It must be a true poisoning in which the blood changes are practically constant and its associated constitutional symptoms should never be lightly regarded. He also points out and emphasizes, in the same article, how the clinical history of uremia may vary from time to time, especially in the intensity of its toxic symptoms and that also when the nervous, the circulatory and the gastro-intestinal systems are apparently gravely affected, that they may, and often do, resume again their normal functions.

Early Diagnosis.

From the foregoing very brief discussion of our present knowledge concerning the etiology of sub-acute uremia, I believe it is at present impossible for us to say exactly what are the earliest symptoms of uremia. These symptoms may be, and probably are, frequently present but their significance and presence combined, as I believe them to be, with those of other clinical syndromes, are of such a nature that they are most difficult to disentangle.

Because of the above and also because an early diagnosis is

essential to early treatment, the chief purpose of this paper is to encourage a more careful and intensive clinical study of this condition. All that we can do at present is to recognize the fact that uremia is, in all probability, very closely associated in various ways with many other conditions, such as chronic nephritis, arteriosclerosis, hypertension, acidosis and other metabolic diseases and conditions. My chief contention is that for a long time these other conditions have been and are now being very carefully studied to the exclusion of the uremic syndrome. I wish particularly to impress upon my listeners my conviction, borne out by a great many years of careful clinical study, that uremia is a much more common condition than we suspect and that its importance to the public demands a much more careful study of its origin, diagnosis and treatment than heretofore it has received. After it has progressed beyond its early stages it is comparatively easy to diagnosis and in its final stages almost anyone can recognize it.

Some of the symptoms I am going to allude to are, as before mentioned, more or less closely associated with other better known and more carefully studied conditions. However, I do not feel that our present knowledge will enable us to disentangle them. My personal experience has taught me to be on the lookout for uremia in all cases of hypertension, especially those closely associated with cardiovascular diseases in which the symptoms of dyspnea, edema and transient attacks of blindness and slight but temporary attacks of dizziness are a feature. Slight attacks of drowsiness are probably early symptoms. Early morning headaches are thought by some to be an early symptom; personally I regard headaches as a rather late symptom. Slight impairment of the sense of touch, slight mental perversions, such as irritability of temper, transient attacks of tremors, cramps and asphasia, especially when associated with the neurasthentic syndrome, may be early symptoms.

Time, of course, in such a brief review of this subject, will not permit me to deal with the subject of symptoms as it deserves, but I will refer those who are particularly interested to the useful clinical classification of uremic symptoms which are mentioned by various authors, principally Barker, who groups these symptoms under four headings:

I.	Digestive System Symptoms	anorexia
		nausea
		vomiting
		hiccough
II.	Circulatory System Symptoms	hypertension
		myocardial insufficiency
III.	Respiratory System Symptoms	dyspnea (uremic asthma)
		dyspepsia
IV.	Skin Symptoms	puritus
		erythema
		papular and other eruptions.

Finally I would stress the great importance in the early diagnosis of uremia of the early use of chemical studies of the blood and urine (blood chemistry and functional urinary tests).

Early Treatment.

If I were asked to associate early diagnosis with early treatment I would sum it up in one word, *elimination in all its forms,* catharsis, diaphoresis, venesection, etc.

Just recently I have had under observation several doubtful cases in which the most marked improvement took place when these patients went away for their summer vacations and visited various health resorts, where they got plenty of rest in the fresh air and an abundant diet of fresh fruit and vegetables, salt rubs, baths and variety of simple hyprotherapeutic and electrotherapeutic measures. Their disease was not recognized nor were any measures taken to study their condition scientifically, but they were treated successfully so that they returned home feeling fine and with few of their former symptoms. Just as soon, however, as they resumed their former habits of life, their former symptoms reappeared, usually within a few months' time.

Conclusions.

I conclusion I would like again to stress my conviction that if any of my audience still have any doubt how very frequently uremia is present, that they can soon convince themselves of the truth of my contention by undertaking a careful study of that large

group of semi-invalids which, among other symptoms, are always complaining of the presence of all sorts of vague aches and pains which come and go. This group, usually individuals over forty, are often grouped clinically as sub-standard cases, neurasthenics, endocrine defectives, etc. Subject these cases to blood chemistry and renal functional tests and you will be surprised how defective you will find their elimination to be. Treat these cases with a careful and sensible course of elimination and you will be surprised to find what brilliant therapeutic results you will obtain.

My final conclusions are as follows:

I. That the use of the term uremia from an etiological standpoint is probably a misnomer, as probably many other organs other than the kidneys are involved in the production of this syndrome, but that it would be unwise at present not to retain this term.

II. That chronic uremia is probably a much more common and frequent condition than is usually supposed, that it is often overlooked, especially in its early and more curable stages.

III. That its metabolic origin should be more generally recognized.

IV. That its great importance as a factor of ill health should justify a more careful and intensive study of its origin.

V. That some disturbances in the function of the liver are probably a very important and common factor in its production.

Note. The author has thus far never studied a case of uremia in which he was satisfied that the liver function was normal, while on the other hand, he has seen many cases of severe kidney disease in which uremia was not a factor.

VI. Finally the prediction is made, that when we finally succeed in determining the as yet unknown toxin which produces uremia (the term toxin is used advisedly as it as yet has not been determined whether a bacterial or a chemical poison is the cause), that it will be found that the same cause which produces uremia will also be a casual factor in the production of many other clinical syndromes with which it is now so often associated.

THE DISADVANTAGES AND LIMITATIONS OF CLINIC OR GROUP MEDICINE.

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The terms group practice and clinic medicine have been applied to quite a number of methods of practice, the main factor in all, however, being that the patient is attended, using the word "attend" in its broadest sense, by a group of physicians rather than one.

For the purposes of this paper I have limited the name "group practice" to what is generally understood as consultation practice, where the patient or his physician seeks other advice than that of his usual medical advisor.

It was the custom of Osler and is the custom of all good internists of today to seek special advice when they deem it necessary, but this advice is sought in each case from the specialist who in the given case can best advise, and not necessarily from the specialist who is allied with the group head, and it is on this fundamental point that group practice differs today from good consultation practice.

In a critical study of any popular movement it is desirable to know its origin and ascertain, if possible, the underlying causes for its beginning and growth.

The successful start of group medicine might be said to date from the recognition of the excellent work done by the Mayo brothers and their associates at Rochester, Minn., such recognition occurring between the years 1905 and 1910.

In trying to ascertain the causes leading up to this radical change in procedure, it is well to try and visualize the conditions of medical learning and education, and the advance in medical and allied sciences during the preceding quarter century.

Medical education in this country was essentially clinical, and showed but moderate advance and improvement from its beginnings in the opening of the nineteenth century up to 1890, at which time the number of schools had increased to a total of 160, the maximum

number ever reached, although the matriculates continued to increase until 1904, in which year over 28,000 students were matriculated. The brilliant work of Pasteur and other scientists had by this time become so generally recognized that it was necessary to entirely recast medical education and give the necessary training in what we term today the scientific branches, which meant that no college could exist on the fees paid by students, and as a result of this, as well as the pressure of an enlightened medical public opinion, the number of colleges have decreased since that date to sixty today which are recognized as giving a satisfactory four year course in medicine. The leading physicians of the country, as a class, at the time of the first broad recognition of the success of the Mayo clinic, were men who had graduated in the period prior to the marked improvement in medical education, and for that reason many of them had only a theoretical knowledge of the scientific advancements, and were, in a measure, incapable of using these advantages to their full opportunity. It was during this quarter century that specialism made its great advance, due not only to the advances in medicine proper during this period, but also due to advances and discoveries in kindred sciences, the Roentgen ray and the electric light being examples. While these advances tended, as a whole, to develop specialism, there were a few which, on the other hand, allowed the general practitioner to encroach upon the specialist's field, such an advance being in the development of the electrical ophthalmoscope, which permits the trained internist of today to know the fundus of the eye as he never knew it, prior to the use of this instrument.

The formation of new specialties, and the growth in all, made it impossible for the internist to keep abreast with this rapid advance in knowledge, but to a far less extent, than is generally supposed, in the diagnostic side as contrasted with the operative or therapeutic.

An even greater factor in the growth of "Group Practice" has been the wide acceptance of the "focal infection" theory of disease, and the development of surgical technique to the point where the immediate dangers from an operation are so slight that frequently an operation is used, like quinine, as a therapeutic test, and like quinine frequently fails to cure the condition.

The history of the growth of the "focal infection" theory is of interest. From the standpoint of therapy it might be said to date from the report of the committee of the British Medical Association on Rheumatic Fever published in 1889, which called attention to the frequency with which rheumatic fever had been preceded or ushered in, by a sore throat. An even earlier paper bearing on this theory was that of Riggs in 1875, in which he called attention to the association of pyorrhoea and "rheumatism," and showed that following the curing of the pyorrhoea the rheumatism had disappeared, and Riggs insisted that the systemic condition was due to the pyorrhoea, and not the reverse, as was generally accepted then. This paper made no impression at the time beyond the giving of a personal name to pyorrhoea.

The report of the British Medical Association committee did, however, bear fruit, and slowly from this time onward the tonsils were recognized as the portal of entry for the organism causing rheumatic fever with its serious complication of carditis, and there resulted the practice of removing the tonsils and thus preventing other attacks.

The wide acceptance of the "focal infection" theory, both by the profession and the public at large, played a large part in the growth of specialism, since it was believed by the public and by the profession that only the specialist was able to decide whether or not a tissue (or organ) was diseased. The ground for this belief on the part of the profession was the vast literature that had accumulated, so vast that no one mind could keep abreast with it, and few realized that this literature had relatively a very small part pertaining to diagnosis, and even that was controversial to a large degree.

Today the difficulty in the diagnosis of the focus of infection, or portal of entry, is not in finding a tissue that is pathological but in deciding whether the lesion is "historical" or healed, or whether it is active. In discussing this point let us take up that small area of the body that accounts for over 90% of "foci" that are treated medically or surgically, namely, the mouth and nose and the structures adjacent. Here the tonsils and teeth are most frequently looked upon as the offending members, and can we tell always, or even frequently, whether a lesion is healed or not? Today laryn-

gologists differ as to the tonsil, some condemning the submerged tonsil, other the hypertrophied, others again those showing adhesions, or atrophic, while some judge by the degree of enlargement of the tonsillar glands. Within the past year I have asked three prominent laryngologists of my home city as to who was the best judge of the need of a tonsillectomy, the laryngologist or the good family physician, and all three unhesitatingly said the internist.

If a tonsillectomy were a minor operaton, this difference of opinion would matter little in most cases, but this is far from being the case, save in young children, and even here the growing list of lung abscesses following tonsillectomy makes one hesitate in saying that there is little or no danger in the operation.

With reference to the teeth there is an equal divergence of opinion. A few years ago any tooth was condemned at once if an x-ray picture showed bone absorption around the apex, but careful scientific work like that reported in the April 1922 number of the Journal of the National Dental Association has taught us that these areas are frequently sterile and remain so, and play no part in metastic disease. I recall vividly two cases in the past bearing on this point. In one, the patient was an extremely good looking middle aged woman suffering from infectious arthritis of the knee, who had been advised by her physician and her dentist to have extracted a second molar and an upper central incisor. She did not mind losing the molar, but objected strongly to parting with the incisor, and consultation was sought, and as a compromise only the molar was extracted, she saving her front tooth, and there was recovery from the arthritis and no return of it in the six years that have elapsed. The other case was that of a young girl, a psychasthenic who had had all of her teeth extracted on account of a mild arthritis, and following which and undoubtedly caused by it, there had developed the mental attitude that made life almost unbearable.

These two cases are cited to show that even extraction of teeth are not always minor events.

The difficulty of diagnosing activity of diseases elsewhere than in the area cited, is on the whole, more difficult than where tissues can be seen, and the paper of Dr. Thos. R. Brown, of Baltimore, in the November 15th, 1919, number of the Journal of the American

Medical Association, on the after effects of operations in the upper abdomen illustrates that diagnostic operations, unlike quinine, are not harmless.

All that I have so far said with reference to "focal infection" would apparently apply with equal force to the consultant of the Osler type as to the head of a diagnostic clinic, save in two important factors. The first factor of importance I have already referred to in contrasting the choice of specialists afforded in one case in getting advice as compared with its lack in the other. An even more important factor is the feeling of individual responsibility that cannot be present to the same degree where the "buck can be so readily passed" as in the diagnostic clinic.

Professions and occupations differ widely in their technique, but have many points of contact where organization is concerned and in that broad twilight zone having to do with "human nature."

Business has learnt through such terms as "soulless corporation" that organization has its penalties, and the trend today in political development is to individualize responsibility as leading to efficiency.

A claim made frequently for the diagnostic clinic is that through the thoroughness of numerous examinations, diagnoses are arrived at that would be unrecognized by the trained diagnostician. This is true in a very small percentage of cases and this percentage depends entirely upon the ability and routine of the consultant as compared with the diagnostic head of the group. If all diagnostic tests caused no discomfort, pain or danger it would undoubtedly increase the percentage of early diagnosis, but I doubt if any clinic or group has adopted as a routine a cystoscopic examination which would from time to time disclose a bladder tumor when no signs or symptoms had arisen to direct attention to its possibility.

A danger today of clinic or group medicine is the doing of unnecessary things at the patient's expense in time, pain, danger or cash expenditure. Sir James Mackenzie has called attention to this in his article entitled "The Future of Medicine"—and says: "Suppose a physician directs these researches, the very fact that he has to call in the help of others implies that he is not able to detect the

phenomena produced by disease, and if he is, he is not capable of doing this, how can he be qualified to assess their value."

The discussion so far has dealt more with what interests the physician, namely diagnosis, as contrasted with what interests the patient, therapy, and there is evidence that group medicine today is leaning to that fallacy of all medical cults, the treating of disease rather than the patient. Anyone who has had experience with good hospitals and sanitaria, both large ones and small ones knows that the therapy is better in the small one through individualism, necessarily lacking to the same degree in a large hospital that must have organization to exist.

In closing I can't help recalling a remark I laughed at some years ago, when a very shrewd countryman surprised me by saying he preferred an elder physician to a younger and better trained man, and gave as his reason that "he knew his constitution." If he had only said psychological reaction I would not have laughed.

PAY CLINICS.

By WALTER L. NILES, M.D.

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On November 1st, 1921, the dispensary of the Cornell University Medical College in New York City, which had previously been operated as a free dispensary, was reorganized on the basis of a pay clinic. It at once attracted considerable newspaper notice, some of which was undesired, and has encountered vigorous opposition from certain elements of the medical profession. The venture was regarded as an experiment in medical sociology, and must still be so considered, but sufficient experience has now been accumulated to warrant a consideration of its accomplishments and an inquiry into the justification or necessity of pay clinics for promotion of the public welfare.

It is evident that a considerable portion of the people cannot secure the best medical advice and treatment under the present organization of the profession. This is appreciated by the public, and has induced a distinct and growing strain in the relations between the public and the medical profession. It is true that only the very rich and the very poor can command the best medical service today, thus leaving the great mass of industrious and ordinarily self-supporting wage-earners to accept incompetent service or charity, for which they have no relish, when serious or prolonged illness overtakes them. Just how large this class is, cannot be determined from the medical standpoint alone, but must be studied by relating the economic status of the family to the amount of sickness and the cost of the necessary medical treatment. It makes a great difference whether a man with a certain income has no dependents or has a large family, and whether illness is trivial and of short duration, or seriously complicated and chronic, requiring extensive investigations and diagnostic consultations. Too often he is obliged to forego the benefits which might be derived from the applications

of modern science. Studies made along this line in New York City by Dr. Michael Davis, Executive Secretary of the Committee on Dispensary Development, led to the conclusion that about one-third of the population are unable to meet the expense of prolonged illness or of care by specialists.

For these reasons the medical profession is faced with the necessity of readjusting its relations to the public; it is forced to relinquish the traditional individualistic status and conform with the advancing social forces of the day. Whatever our desires, we are compelled to change, for the very good reason that we are servants of the public, and it is not getting from our profession what it believes it should get. It is possible, I believe, to satisfy this demand without any greater expenditure of time and energy on the part of the profession by a readjustment of the efforts now made to prevent and relieve the ills of mankind.

The factors which have brought about this state of affairs are numerous, but the principal one is the great expansion of the field of knowledge in those sciences which are fundamental to medicine. It is no longer possible for one man to be conversant with, and skilled in, all of the methods essential for a complete diagnosis in a considerable percentage of cases, particularly the chronic conditions, and to assure a high quality of service various specialists must be consulted and extensive laboratory investigations must be made. These are costly and, under our present system of individual practice, often prohibitive. The public is however insistent upon securing the best quality of service at a price which can be met by those of an average economic state. The remedy lies in better organization of the components of the medical profession; a transition from individual practice to co-ordinated practice.

The decision to reorganize the Cornell Clinic on a basis whereby the patients pay a moderate fee and the physicians are compensated for their services was the outcome of a survey of the dispensary situation in New York City made by the Public Health Committee of the New York Academy of Medicine, which recommended the establishment of pay clinics. It was thought that a dispensary governed by a medical college or hospital and in which active teach-

ing is being conducted, would afford the best test for the experiment. In reorganizing the clinic the College had two purposes in mind:

1. To improve the facilities for instruction of students and for scientific research available to the students and faculty of the College.

2. To assist a large class of persons, who it is believed exist in the community, who can pay something for medical services but not the rates usually charged in private offices, particularly when the care of specialists or expensive diagnostic work is required.

In developing the organization, certain aims have been held clearly in view. We endeavor to give a high quality of service to the patients by providing an adequate professional staff which is paid for the service rendered, and by adjusting the number of patients admitted in each department to the staff and physical equipment so as to secure individual attention for each patient. A second important principle is the provision of sufficient non-medical assistance so that the time of the physicians may be conserved for strictly medical work. This includes nurses, clerks, stenographers, technicians and executive assistants, all trained persons, for the detailed clinic management and social service. As a third principle, it is the endeavor so to adjust the relations between the different departments as to promote team work and co-ordinated study of the patients as a whole. There is a central record system, all of the data regarding each patient being filed in one folder and indexed.

It is the aim to also provide the physicians with sufficient knowledge regarding the personality and home environment of each patient to enable advice to be adapted to the individual needs and to develop adequate methods for follow-up and for the study of end results.

All of the usual departments operate simultaneously, and numerous special classes have been organized. Five afternoon and two evening clinics are held weekly. These requirements necessitate a large staff, eighty physicians holding regular paid appointments besides thirty provisional assistants not receiving pay. There are seventy-eight persons on the non-medical roll including registrars, clinic executives, nurses, historians, pharmacists, technicians, etc.

The clinic is under the direct supervision and control of a

committee of the faculty headed by the Dean. An executive officer, or clinic director, has immediate direction of the administration. The teaching schedule brings the senior faculty to the clinic and the clinic chiefs and many of the assistants are also members of the teaching staff. Several heads of departments are finding that the clinic is more attractive than the hospital wards and attend regularly.

The general admission fee for examination or treatment is $1.00, but additional charges are made for extensive laboratory and x-ray examinations, minor operations and prescriptions. In the diagnostic clinic, which receives patients referred in writing by physicians, each patient is given a thorough general examination in the department of medicine with all necessary examinations and consultations with other departments and a considerable amount of routine laboratory work for $10.00. Extensive x-ray and other laboratory investigations beyond the routine are extra. A written report is rendered to the referring physician by the chief of the medical clinic, and no patients thus referred are given treatment except at the request of the referring physician.

The policy governing the admission of patients has been carefully worked out with the aid of the staff of the Committee on Dispensary Development (of the United Hospital Fund) and may be summarized as follows:

Patients who need service which they could not otherwise secure within their means will be accepted; others will be referred to private physicians, hospitals or free dispensaries. The relation of income and responsibilities (estimated on an annual basis) to the cost of the needed service will be the basis for decision. Patients about whom there is a doubt as to the eligibility for admission may be accepted temporarily, pending further investigation. The clinic physicians are requested to call the attention of the director to any instances in which they think a mistake has been made by the admission department. Patients sent by private physicians will in general be accepted without question, although the clinic reserves the right to investigate and reject such cases. Applicants who are referred to private physicians are given the names of at least two selected from a list which has been made up from the staffs of representative hospitals and dispensaries.

From the opening of the pay clinic the number of applicants has exceeded the facilities, but better and larger organization together with an increasing proportion of return visits has increased the average attendance per session from 240 in November, to 336 in March, which approaches the maximum number for which efficient service can be rendered.

The cost of operating such a clinic is very large, averaging $16,500 per month, not including the overhead expense of the college administration which is not charged against the clinic. The deficit has averaged $3,500.00 per month, but is steadily diminishing, and it is hoped it will soon amount to little, not including, however, $20,204.88 which has been expended for equipment, alterations, etc. The venture has been possible only by generous support of the Dispensary Development Committee of the United Hospital Fund which has agreed to meet the deficit during the initial period in excess of the former deficit of the dispensary as maintained by Cornell on the old basis.

Certain studies of the first five months of operation reveal interesting facts. Five hundred consecutive new cases were reviewed, and it was found that over 80% were eventually assigned to other departments than general medicine and surgery, and more than half of the remainder required examinations in special departments; that is, 90% of the patients require a specialist's services. The same survey showed that only 10.8% were suffering with acute illnesses. 86.6% were classed as chronic, and 60% stated that their trouble had existed one year or more.

The question naturally arises whether such a clinic should confine itself to diagnostic work, or give treatment as well. It appears likely that diagnostic groups may render valuable service to those who are able to follow out the recommendations for treatment under the direction of competent general practitioners or specialists. I doubt however if the diagnostic clinic goes far enough in meeting the needs of the class of people for which the pay clinic is designed. The establishment of a diagnosis is of little service unless the necessary treatment can be given subsequently. Having this in mind, an attempt was made to estimate the probable duration of treatment in the series surveyed with the following results:

Treatment needed for less than one month	20%
Treatment deemed necessary for an indefinite period	63%
Operation advised	11%
Undetermined	6%

Therefore, it appears evident that if we confine our work to diagnosis, we will but partly meet the needs of a large class of patients.

For the purpose of judging the efficiency with which the admission system is conducted, a special study was made by independent investigators who visited eighty patients in their homes and places of work. One case out of eighty was believed able to pay a private physician, and therefore wrongly admitted. On the other hand 25% were found to be less well off than the registrar had judged at the admission desk.

A review of the first five months of operation on the new basis shows that so far as the college is concerned the clinic is functioning very satisfactorily. The volume of teaching material has vastly increased and the proportion of interesting cases is larger. No more difficulty is encountered in utilizing the patients for demonstration than before. A very competent professional staff has been readily recruited and the physicians agree that the method of operation gives them more instructive opportunities. The college can now attract its graduates to the clinic where they have every advantage for pursuing clinical investigations in co-operation with the laboratories under competent supervision. This practically amounts to fellowship in graduate work and diverts them from paid positions in industrial concerns where they generally remain sterile.

There is a large demand for such co-ordinated service on the part of the public. Only ten per cent of the applicants present acute conditions, and ninety per cent require the services of one or more specialists and it is believed that few of them could otherwise be efficiently served. The clinic is certainly rendering better service than was given under the old plan.

In considering the relation of the profession it appears evident that few individual physicians can give honest service for the fees which we charge. Inefficient service should not be countenanced at any price. Very few dollars are diverted from the conscientious

doctor's pocket; on the other hand, we pay $7,500 monthly in salaries, very little of which would otherwise reach the profession. If our economic standards are correctly formulated and our admission system is efficient, as it now appears to be, the patients are not being duly burdened financially, neither are they pauperized, thus maintaining their dignity and self-respect.

Clinics which are designed for the benefit of the great mass of wage-earners should not and cannot be conducted for profit, and must therefore be maintained by established organizations such as hospitals or medical schools. Such clinics are needed to meet the demands of a considerable part of the public, and I believe that by satisfying it in this manner, the state will be less likely to interfere with the medical profession and practitioners of the cults will find their fields of activity contracted.

DISCUSSION.

Dr. Minor: I notice in the doctor's paper (I can not quote his words) that he takes it for granted that the treatment the patient will get outside of the diagnostic clinics is necessarily inefficient. If that is so, it is an indictment of our medical school of which we are so proud.

Has the medical school lost its ability to educate a man who is fit for practice? Is the general practitioner hopelessly a thing of the past? If so, God help the country! In New York it may be possible, but God help the country. It is up to the educators of the country to find some means by which they can educate a man who is competent to take care of his patients.

I am not a neurologist, and some other man might not know as much as I think I do about lungs, but we have got to realize that the bulk of the practice in this country has got to be done by men who cannot get all of this assistance, and the general practitioner needs a lot of sympathy.

I recognize that things in New York are different, and I recognize that a man coming to a Cornell clinic has certain advantages, but we have heard them tell us, who have gone through a group system, that they felt they had been through a machine and had not come in contact with a man.

Another point which makes it difficult is the history. I think the idea of letting the history be taken in the front office is rot. No man gets a proper idea of history unless he takes it himself. If you get your reports from ten or fifteen specialists, and try to co-ordinate them, it is going to be difficult.

I recognize that it is a difficult thing to work out. The work Dr. Niles is doing is opening a new way. I hope to God that our practice has got to go into those lines to make a success. You are talking of the good of your patient —you have no business to talk of the effect upon the student, but the effect upon your patient.

I recognize that you will get many things, but I am very far from being ready to accept the fact that the solution you offer is applicable or successful anywhere except possibly in just such a situation as you tried to apply it.

Have the young men all over the country got to think that they cannot practice medicine? A man has got to have faith in his ability to help his patient, and if he has got to run around and get a lot of men to help him, his patient is in a bad way. I have no solution. It is a question of the education of our doctors right in the beginning. Is it possible today, with the development of medicine we see going on all around us, that those of us are trying to do the best we can, and we cannot keep up with it? Can we not turn out a doctor who can help his patient?

THE PRESIDENT: Is there further discussion of these papers?

DR. SWAN: I am sorry I did not hear these papers. What Dr. Minor said about the importance of the physician taking the history of the patient I heartily agree with. I think we all have found that where some one else took the history for us we have lost personal contact and personal confidence, and a good many items of interest are left out which were important, and we had to get them afterwards. I think there should be personal contact with a patient.

One thing further with regard to focal infection of the teeth. That is one of the difficult and important things that we have to deal with in general medicine. I am referring to patients with tuberculosis.

I think it is frequently the case that we have patients with tuberculosis who are doing badly, and under proper regime faithfully carried out, sometimes it is quite frequently the situation that tuberculosis clears up and the patient improves after the extraction of some diseased teeth. The difficulty is in deciding what teeth should come out.

I think we have got to realize that the negative result of an x-ray picture dose not mean that the teeth are not diseased; on the other hand a positive result does not always mean that a tooth is diseased or a dangerous or important infection.

Sorting and deciding in an individual case what teeth should be extracted is an important thing, because the extraction of a tooth is by no means an unimportant, trivial matter.

THE PRESIDENT: Is there a further discussion of these matters?

DR. MERCUR: I would like to say to Dr. Minor that my idea of pay clinics is that their use should be confined to a particular set of cases.

Let me explain what I mean. I, for one, am a believer that the ordinary well trained practitioner that is being turned out in our medical schools today can easily diagnose and can easily treat within half an hour or an hour, and take a history of from eighty to eighty-five per cent of the cases that come to him; in other words, there are only fifteen or twenty per cent of cases that the ordinary man is puzzled about. Those cases require a very unusual, long continued history. They require the use of very elaborate and time-consuming laboratory tests, and they require unusual skill.

In olden times the general practitioner thought he diagnosed all of them. Fifteen per cent were undiagnosed, and died by the grace of God. The general man knows today that fifteen per cent of the cases, if he is a good practitioner, he has to refer to specialists. In other words, that fifteen per cent of cases, refers to a group of men, specialists, who have became specialists just to do that work.

Only ten per cent of the people of the country can afford to employ a particular specialist for those cases, sixty per cent of the people of the country, self-respecting American citizens of moderate means, who are not in the prince class, we have no place to send them, and that is my contention, that a pay or diagnostic clinic for people of moderate means who are not at the top or at the bottom, ought to go where they can have a fixed fee, get those services which their physicians cannot give them, and now have no place to send them.

Dr. Niles: Dr. Wilson and I have different classes of people in mind. All the dispensaries in New York City are charging a fee of twenty-five cents per visit, and some fifty cents, and we are not the only clinic charging a dollar. It is our intention to give a specialized service for this fifteen per cent of otherwise undiagnosed cases. People who otherwise would not be able to secure such service can now get it, and people ordinarily self-supporting, when they have a prolonged or difficult or complicated illness, cannot get proper service.

Referring to Dr. Minor's remarks, I will say that the sole reason that Cornell established this clinic was an attempt to turn out better doctors. Our dispensary was good, and averaged well with other dispensaries in the cities, but in New York and many other cities the dispensary situation is deplorable. It does not compare with that in hospitals by any means. One of the reasons is the lack of facilities and difficulty in getting competent men to work in dispensaries. Formerly masters of the profession worked there, but today they very seldom do. We have improved our dispensary, and also the dispensary teaching, and we can turn out more efficient doctors. The general practitioner is going to be the chief of the diagnostic group.

Dr. Minor: How about history?

Dr. Niles: We allow forty-five minutes to every new patient.

Dr. Minor: Who takes that history?

Dr. Niles: The regular physician. He takes the history invariably.

Dr. Wilson: In regard to history: About a year ago I asked some men which they would prefer, a physical examination or a history, and these men said they would prefer a physical examination. I think this is very often neglected, and two weeks ago a man, well known to all of us, was telling me about a case that was sent to him. This man had had everything done to him—the only things that had not been done was a thorough history and a thorough examination.

Dr. Darlington: I would like to say one thing. I happen to be the treasurer of the New York College of Dentistry. When we came from the war

we had no money, so I increased the fees a little bit at the pay clinic, as we needed the money for scientific apparatus for the students, and this year, up to the last week, we had made a profit of $51,000, which we are turning into scientific apparatus for the students, so that will take care of a lot of apparatus which the students need.